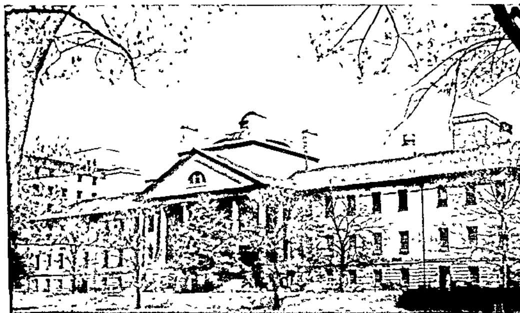
PHYSICAL



The Bulfinch Building, Massachusetts General Hospital, erected 1821.

THE WILLIAMS &

DIAGNOSIS

By F. DENNETTE ADAMS, M.D.

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FOURTEENTH EDITION

WILKINS CO.



Baltimore • 1958

BY RICHARD C CABOT, M D
Late Professor of Clinical Medicine, Harvard University, and Chief of the West Medical Service, Massachusetts General Hospital

First Edition, 1900, Second Edition, 1903,
Third Edition, 1905, Fourth Edition, 1909,
Fifth Edition 1912, Sixth Edition, 1915,
Seventh Edition, 1919, Eighth Edition, 1923,
Ninth Edition, 1927, Tenth Edition 1930,
Eleventh Edition, 1934

BY RICHARD C CABOT, M D AND
F DENNETTE ADAMS, M D
Twelfth Edition, 1938

BY F DENNETTE ADAMS, M D
Thirteenth Edition, 1942
Fourteenth Edition, 1958

Library of Congress
Catalogue card number
58-7891

Composed and printed by WAVERLY PRESS, INC.
Mt Royal & Guilford Aves, Baltimore 2, Maryland, U S A.

To my daughters

JILL *and* ELLIOTT

medicine to their father's mind and heart

Publisher's Foreword

Physical Diagnosis, by Dr Richard C. Cabot, was first published in 1900 and went through eleven editions in thirty-four years In 1937, when a more complete revision than any previously published was required, Dr. Cabot, who by then had largely withdrawn from medicine to devote himself to the study and teaching of ethics and sociology, requested Dr F. Dennette Adams to undertake the task

In the twelfth and succeeding editions, Dr Adams has so completely revised, rewritten, and reorganized *Physical Diagnosis* that it is by now as uniquely a product of his own learning as the first eleven editions were of Dr Cabot's

The alteration of the title page of this fourteenth edition so that it bears evidence, as does the text, of the complete change in authorship, seems therefore to be demanded by the realities of the situation The work is not less, for that reason, a monument to Dr Cabot's memory In recognition of this, we can conceive no more fitting tribute than the one which appears on the facing page—an excerpt from Dr. Adams' preface to the thirteenth edition

Richard C. Cabot, M.D.

The death of Richard C. Cabot on May 7, 1939, cost American medicine a gifted teacher and great leader. Students and associates who knew him at the Harvard Medical School and the Massachusetts General Hospital—indeed all who were privileged to know him—felt the impact of his vigorous intellect and the ardor of his lofty spirit. He was a distinguished practitioner, a zealous pioneer in medicine, and a sympathetic, outspoken champion of the underprivileged.

To the practice of medicine Richard Cabot made four outstanding contributions. He was the first adequately to demonstrate and emphasize the importance in diagnosis of microscopic and serologic study of the blood. In 1896, only four years after his graduation from the Harvard Medical School, he published *Clinical Examination of the Blood* and three years later, *Serum Diagnosis of Disease*.

Diagnosis and its teaching again benefited when, in 1906, he introduced at the Massachusetts General Hospital the Clinico-pathological Conference, which at once became and has remained an integral and vitally important part of the hospital's program. It has since been adopted in many medical schools and hospitals elsewhere.

In a paper published in the Journal of the American Medical Association in 1914, he made his third significant contribution: the placing, for the first time, of proper emphasis on the etiologic diagnosis of heart disease.

What Dr. Cabot himself would have reckoned his greatest achievement was made at the turn of the century when he was physician to the Out-Patient Department. Here he saw repeatedly demonstrated the futility of treating, on a purely medical basis, the patient who needed financial help and counsel in social problems as imperatively as he needed medical advice. With characteristic initiative he engaged, partly at his own expense, a social worker. From this small beginning, under his guidance and that of Miss Ida M. Cannon, grew the Social Service Department—now one of the hospital's most important activities. It has served as a model for similar departments in hospitals the world over.

F. DENNETTE ADAMS, M.D.

Preface

The literary heritage of such a book as *Physical Diagnosis* would seem to entail an obligation to continue at least the plan established by the original author, Dr. Cabot In spirit this volume does so, though the circumstances noted *below have involved such considerable* changes in form and content that few recognizable features of the first text now appear However, Dr Cabot would, I believe, readily acknowledge this edition, the fourteenth, as the lineal descendant of his pioneer work of 1900.

In the first eleven editions, Dr Cabot followed the then valid plan of describing only those techniques which he himself thought valuable, and of discussing only those aspects of disease with which he had personal acquaintance But the tremendous growth of medicine in the first third of this century had made it obvious to him that no one person's experience could be sufficient to include all that the student of physical diagnosis should know For this reason, when he asked me to undertake preparation of the twelfth edition, we agreed that it would be necessary not only to enlarge and reorganize the text but also to supplement our own knowledge by drawing freely on the experience of others This decision has continued to be the guiding principle for the subsequent editions of *Physical Diagnosis.*

Since the last edition, in 1912, medicine has progressed so rapidly and diversely that, in some areas of diagnosis, emphasis has changed radically, procedures once important are now less so, while others have been newly developed or accentuated in value Increased knowledge of certain maladies has required extensive rewriting of their descriptions; others have demanded more detailed discussion than was formerly pertinent The development of unipolar leads in electrocardiography has necessitated complete revision of all material on that subject Improved techniques and greater interpretative acumen in radiology have made it advisable to include more x-ray descriptions and illustrative films

Successive editions of such a book as this stand as evidence to the status of man's understanding of disease, much as the rings on a fallen tree testify to the wet and dry years, as well as to the primary record of growth The reader familiar with previous editions of *Physical Diagnosis* will observe that generous use of both blue pencil and fresh ink have recorded our increasing knowledge of the bodily processes and our greater ability to interpret their failings

Despite extensive reorganization and revision, the purpose of the book remains the same to show how the patient should be examined, to describe the normal findings, to explain and interpret the important symptoms and signs created by injury or disease Entities are discussed because of my continued conviction that any subjective or objective variant can be effectively taught, understandingly learned, and intelligently appraised only when it is studied

in relation to, not apart from, the circumstances under which it occurs. No effort has been made to cover the entire field of medicine—in fact my aim has been to select only those disturbances that one is likely to encounter from day to day. Both in text and illustrations the unusual and the extreme have been purposely omitted.

Since an appreciable amount of material in the following pages has been brought forward from the twelfth and thirteenth editions, I again offer my thanks, recorded therein, to the many friends who so generously contributed to their preparation It is a source of regret that restriction of space prevents republication of their names

In organizing the new material, I have again drawn freely on the wisdom of many confrères at the Massachusetts General Hospital and of friends in other medical centers whose specialized knowledge has enabled me to write with greater authenticity It is impossible for me adequately to express my appreciation to those distinguished members of the profession who with kindness, patience, and generosity took time from their own many obligations to brief me on important details in their particular fields, constructively to criticize manuscript, and otherwise enable me to profit from their experience and judgment Except where indicated, those listed below are colleagues on the staff of the Massachusetts General Hospital.

INTERNAL MEDICINE *General*—Drs. Leonard W Cronkhite, Jr, Bernard M. Jacobson, Rita M. Kelley, Madan M Singh (Former Resident in Medicine), Maurice B Strauss (Boston V. A Hospital); *Bronchopulmonary Diseases*—Drs Kenneth T Bird, Carl Gardner (formerly Chief T B Section, Cushing V A. Hospital), Donald S King, *Cardiology*—Drs Fred Alexander (formerly Cardiac Research Fellow), Edward F. Bland, Morton G Brown (Lemuel Shattuck Hospital, Mass Dept. of Public Health), Allan L Friedlich, Benedict F. Massell (House of The Good Samaritan), Howard B Sprague, Edwin O Wheeler, Paul D White, Conger Williams, *Endocrinology*—Drs Joseph C Aub, Fred Bartter (formerly Clinical Associate in Medicine), Anne P Forbes, Philip H Henneman, Farahe Maloof, James H Means, John B Stanbury, *Gastroenterology*—Drs Perry J Culver, Chester M Jones, Roger S Mitchell (Colorado Foundation For Research In Tuberculosis); *Infectious Diseases*—Drs John C. Snyder (Harvard School of Public Health), Louis Weinstein, *Nephrology*—Dr Alexander Leaf; *Occupational Diseases*—Dr. Harriet L Hardy, *Rheumatology*—Drs Walter Bauer, Marian W Ropes, Charles L Short, *Syphilology*—Dr. Nicholas J Fiumara.

SURGERY *General*—Drs Marshall K Bartlett, Ernest M. Daland, Robert R Linton, Leland S McKittrick, the late Ira T. Nathanson, Fiorindo A Simeone, Richard H. Sweet, Grantley W. Taylor, Frank C Wheelock, Jr; *Gynecology*—Dr. Joe V. Meigs, *Neurosurgery*—Dr H Thomas Ballantine, Jr; *Orthopedics*—Drs Joseph S. Barr, Edwin F Cave, Eugene E Record, Carter R. Rowe, *Stomatology*—Dr. David Weisberger, *Urology*—Drs. Sylvester B Kelley, Wyland F. Leadbetter, Lorande M Woodruff

DERMATOLOGY. Dr. Jacob H. Swartz.

NEUROLOGY: Drs Raymond D. Adams, Mandel E Cohen, Charles S Kubik, Vincent P Perlo

OBSTETRICS. Dr A Gordon Gauld

OPHTHALMOLOGY Drs Thomas Cavanaugh, David G Cogan, Edwin B. Dunphy, Merrill J. King

OTOLARYNGOLOGY Drs Charles I. Johnson, George F Reed, LeRoy A Schall, the late Harold G Tobey.

PATHOLOGY: Drs Benjamin Castleman, Robert E. Scully, Austin L. Vickery, Jr

RADIOLOGY Drs Jack R Dreyfuss, Joseph Hanelin, Laurence L Robbins, Richard Schatzki, Stanley M. Wyman.

Dr. Daniel Holzman, Cardiologist and Assistant Chief of Medicine, Boston Veterans Administration Hospital, at great expenditure of his time, aided me in rewriting the chapter on electrocardiography and describing electrocardiographic variants discussed elsewhere Dr John C Dalton spent many hours selecting and titling the electrocardiograms reproduced Most of the tracings were borrowed from the Cardiac Unit of the Massachusetts General Hospital and from the private files of Drs White, Bland and Friedlich, they were prepared for publication by Miss Louise Wheeler, Executive Secretary of the Cardiac Unit

Dr. William H Timberlake generously contributed a great deal of time and energy to help expand and rewrite the chapters on neurology

Dr James A Petersen selected most of the x-ray films after a time-consuming and diligent search for those most suitable for our purposes He also assisted in composing their legends Except where otherwise credited, the films reproduced were obtained from the files of the Massachusetts General Hospital

Dr Frederick R Taylor of High Point, N C, having carefully reviewed the thirteenth edition, provided me with many valuable suggestions used in the following pages

If I have failed to acknowledge the kind offices of any other friends, my profound apologies are extended for the oversight

Mrs Muriel McLatchie Miller, Director of the Unit of Medical Art and School of Illustration of the Hospital, and her associate Mrs Edith Tagrin supervised preparation of the new diagrams which were drawn by Miss Sylvia Collard, a recent graduate of the School Mrs. Tagrin also lettered and otherwise prepared most of the illustrations for publication

Many of the photographs came from the Hospital and from the author's files. Those otherwise obtained are acknowledged individually in the legends All reproductions were made in the Medical Photography Unit of the Hospital I am indebted to Mr Donald C Withee, Director, and his assistants Mr Frank T McCarthy, Mr Stanley M Benett and Miss Dorthy Garbardi, for the care and skill with which this work was accomplished The photograph of the Bulfinch Building, which appears on the title page, was taken for the purpose by Mr. Withee.

To the secretaries named below I owe a decided debt of thanks for their

patience, proficiency, and dedication to this work. Mrs Margaret Burnett Sawyer helped in the writing of most of the manuscript; Mrs Shirley Grant Gray assisted with the last four chapters and organization of nearly all of the illustrations, Mrs. Dorothy Wright Sullivan and Mrs. Dorothy Melvin McNeely worked with me on the illustrations and the exacting task of proof-reading. Appreciation is also extended to Mrs Jane Rydholm Olsen and Mrs Mary DeFranco Casey who maintained a smoothly-running office while I was largely engaged in preparing this edition.

F. DENNETTE ADAMS, M.D

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"To study the phenomena of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all. . . ."

—SIR WILLIAM OSLER

1

THE HISTORY

Disease manifests itself by abnormal sensations and events (*symptoms*), and by changes in structure or function (*signs*) Symptoms, being subjective, must be described by the patient. Signs are objective, and these the physician discovers by means of physical examination, laboratory studies, and special methods of investigation such as x ray, electrocardiography and basal metabolism tests

The information thus obtained is of little value unless the physician is familiar enough with the ways in which various maladies affect the body to be able to select and correlate the pertinent facts in any given case and to recognize the clinical picture which they indicate If some of the findings prove irreconcilable with a particular diagnosis, he must be able to judge whether they can be rejected as irrelevant or are important enough to form the basis of a different diagnosis

How thorough the investigation of any case should be is in itself a problem Theoretically, every patient *should* have the benefit of every available method of study Practically, this is often neither necessary nor financially feasible Each patient must be individually evaluated, the physician must decide which tests are advisable and which can safely be omitted Usually the indications are clear. Many simple ailments can be diagnosed without elaborate study In other disorders the patient is too ill for anything but measures to meet the immediate emergency. Should evidence of some unrelated disturbance be found incidentally, further study can be postponed until after recovery from the acute episode, unless there is reason to believe that delay will endanger the patient's life

On the other hand, many cases require thorough study The single symptom, *recent loss of weight*, for example, calls for immediate and careful investigation *Any one of a host of diverse disorders might be the cause: pulmonary* tuberculosis, cancer, diabetes, thyrotoxicosis, or even worry

Sometimes intensive investigation is not immediately essential, although later developments may make it necessary Let us take a case of chronic indigestion Here a detailed history, physical examination and simple laboratory tests are required at the start If this initial survey points to improper diet or eating habits, chronic fatigue or worry as the cause of the trouble, the effect of a proper diet and adequate rest might justifiably be determined before submitting the patient to the trouble and expense of more elaborate study. If after a definite interval of time these simpler therapeutic measures fail to bring relief, further

investigation by means of x-ray, gastric analysis and other special examinations must be made to exclude gastric carcinoma, gallbladder disease, or some equally serious cause

No hard and fast rule dicates how complete the investigation of any given case should be But it is always safer to be too thorough than to run the risk of being too superficial *Mistakes are just as often caused by lack of thoroughness as by lack of knowledge;* by failure to exclude early tuberculosis as a cause of cough, or gastric carcinoma as a cause of indigestion, as by failure to recognize the relationship between some such simple symptom and serious organic disease

TAKING THE HISTORY

The history is the key to diagnosis. Being a record of the patient's story and symptoms, it immediately suggests certain diagnostic possibilities, excludes others, and points the direction which further investigation should take. Sometimes it actually provides the only evidence on which diagnosis can be made. The only manifestations of angina pectoris, for example, may be subjective, physical, laboratory and special examinations may show no variation from the normal. Diagnosis of such unrelated conditions as migraine, gallstones or ulcer of the duodenum sometimes depends much more on an understanding of the patient's story and symptoms and on knowledge of what diseases will produce them than on what can be learned objectively *More errors in diagnosis are traceable to lack of acumen in eliciting or interpreting symptoms than have ever been caused by failure to hear a murmur, feel a mass or take an electrocardiogram*

Taking a satisfactory history is no simple task An expert knowledge of the symptoms which different diseases produce is only one of the requirements Equally important are tact and diplomacy, an understanding and sympathetic manner, a quick and accurate appraisal of personality pattern and an approach that will put the patient at ease. If these qualities are lacking or there is any show of haste, intolerance or irritability, the patient will be so frightened or antagonized that his statements may be hurried, confused or inexact

Use of a stereotyped form is inadvisable, except as a general guide The patient is likely to get the impression that personal interest in his problem is lacking. As a result he will give mechanical replies stripped of incidental comments and volunteered information which so often provide the richest clues Moreover, by limiting himself to a printed form the examiner may fail to follow up the leads he elicits A history must be taken in privacy Presence of a third person may, by embarrassing the patient, make him reluctant to talk freely.

The first facts recorded are the patient's name, residence, age, sex, race, marital state, nationality and occupation. Next he is asked to tell in a few words the nature and duration of the symptoms that brought him to the physician (*Chief Complaint*) Detailed information is then obtained concerning, (1) current symptoms (*Present Illness*), (2) personal life and such emotional factors as may influence it (*Social History*); (3) occupation (*Occupational History*), (4)

daily routine and its associated stresses and strains (*Habits*); (5) past life, especially previous illnesses and experiences having a possible bearing on the present illness (*Past History*); (6) illnesses [illegible]
if and when the patient was in [illegible]
of parents and siblings, particul[illegible]
heritable tendencies or diseases, and communicable diseases (*Family History*); (8) health of wife or husband and children, again with emphasis on transmissible or heritable disorders (*Marital History*)

The standard form for recording a history marshals the facts in chronologic order, the Family History first and Present Illness last, thus enabling the physician to view the course of events in their proper perspective But when taking the history one begins with the Present Illness· first, because the patient's interest is naturally centered on his current symptoms; secondly, because investigation of the other aspects of history can be more intelligently pursued on the basis of clues provided by recent developments Social History should be taken toward the end of the interview By this time the patient should be more at ease, have more confidence in the physician, and be less reluctant to discuss particulars of his private life.

PRESENT ILLNESS

Here one elicits a detailed account of the patient's symptoms and the circumstances attending their appearance Without guidance the patient cannot give all the pertinent facts They must be extracted by careful and intelligent questioning Sympathetic comment is sometimes necessary to encourage the reserved or inarticulate. The garrulous or digressive individual requires kind but firm maneuvering back to the point and the evasive or not wholly dependable one must be questioned closely and persistently

Since the average layman describes his ailments in notoriously inexact terms, he must be closely questioned to determine the nature of what, for example, he calls his "liver trouble", "kidney trouble", or "pain" Pain in one case may mean slight distress, in another a dull ache, in a third, agonizing colic "Dizziness" will be used by one patient to mean lightheadedness, by another, clouded sensorium. Actually, dizziness or giddiness is an abnormal sensation of unsteadiness or of motion within the head; vertigo is a feeling of rotating in space or of rotating surroundings Another much abused term is "gas" A person may incorrectly use this term to indicate any one of a variety of sensations such as substernal oppression or tightness, abdominal distention or rumbling, belching, flatus, epigastric ache or abdominal cramps Only meticulous questioning will bring out the actual nature of the complaint

Careful judgment must be used in evaluating statements which are the patient's own deductions or interpretations For instance, because his pain immediately follows a meal a patient may decide that he has stomach trouble. Actually he may have angina pectoris and the precipitating factor may be not so much the effect of the meal as of the exertion attendant upon climbing a flight of stairs immediately after eating

But even the most rambling and inexact account has some value; it provides a number of useful leads and gives an index of the subject's mental and emotional make-up—no small asset in evaluating the importance of his statements

When the patient has finished his story, the leads it furnishes should be followed up by direct interrogation. This requires a knowledge of just how disease of an organ will affect its function as well as its structure, how one organ can be disturbed by disease in another, how the health of the body as a whole will suffer, what the characteristic subjective manifestations are and how they are related to one another *Leading questions must be avoided*—most patients have a tendency to give the reply which they think the doctor expects. When the case is simple, the facts are usually clean-cut, the implications clear, and a few well-directed questions suffice It is the complex or obscure problem which requires the more skilful questioning

The account usually suggests a number of possible diagnoses To narrow the field, each important symptom is analyzed in the light of these possibilities Diagnoses with which any important symptom proves incompatible are automatically eliminated So are those which are characterized by some key symptom missing from the history *A patient's failure to mention a symptom is no proof that it has not occurred*, it can be excluded only by careful direct questioning.

Because the characteristics of most fundamental symptoms, for instance, *pain, nausea, dizziness* and *dyspnea* vary so widely in the different disorders in which they occur, we must learn as much as we can about any abnormal sensation the patient describes. What is its location, intensity, quality? How does it start and stop? It is intermittent or continuous? How long has it been going on? How is it aggravated or relieved? How is it related to other symptoms, and to bodily functions such as eating, defecation, exertion, sleep and catamenia?

Let us take *pain* as an example Pain in any particular area immediately focuses suspicion on the organs in the vicinity, although it may be referred elsewhere Disturbance in the esophagus causes substernal pain, in the stomach or duodenum, epigastric pain; in the small intestine, pain in midline near the umbilicus. Pain caused by disturbance in the cecum is referred to the epigastrium or to McBurney's point; in the ascending colon, to the lower abdomen just to the right of midline, in the transverse and descending colon, to the lower abdomen a little to the left of midline, in the rectosigmoid, to the left iliac region.

Pleural pain usually appears in the lower thorax but may be referred to the abdomen. When it originates in the diaphragmatic pleura, it may be felt in the upper part of the abdomen or in the neck or shoulder Cardiac pain is usually substernal; it may appear in the shoulders, neck or arms Gallbladder pain is felt in the epigastrium or right upper quadrant and is often referred to the right scapula or elsewhere. Kidney pain usually occurs in the costovertebral area, but renal colic may extend to the flank, the genitals or down the inner aspect of the homolateral thigh.

We have no way of measuring pain. But we can roughly gauge its intensity by finding out whether it makes the sufferer double up, press on the affected

region, cry out, rouse from a sound sleep or require an opiate. These criteria are not absolute They must be considered in the light of the physician's estimate of the patient's *threshold of discomfort: What is severe pain to a sensitive, nervous or exhausted person might be mere discomfort to one who is more phlegmatic or robust*

Colicky pain suggests disturbance of a hollow viscus, brought about by overactivity of its musculature. The colic of stone in the ureter or common duct is usually sharper and the spasm shorter than in uterine or intestinal disorders Constant, boring pain is often the result of pressure, ulceration, or invasion Recurrent sharp, swift stabs of pain suggest irritation of a sensory nerve or its root. The typical pain of angina pectoris is oppressive or constrictive

The nature of a disorder is often suggested by the manner in which pain begins or ends and by its duration Angina pectoris starts and ends abruptly and lasts only a few moments; the pain of myocardial infarction begins abruptly but lasts hours or days and subsides gradually. In neurocirculatory asthenia a prolonged precordial ache is described, less often a series of momentary stabbing sensations An acutely infected kidney will cause a dull pain of long duration, whereas that of renal infarct begins abruptly and soon disappears Peptic ulcer is characterized by intermittency of pain, which waxes and wanes from month to month or year to year, and when present varies from hour to hour depending in large measure on the amount of food in the stomach The pain of gastric *carcinoma, on the other hand, usually begins insidiously, but once established* it is quite continuous, although it may fluctuate to some extent

Light can often be thrown on a problem by the patient's description of what alleviates or aggravates his distress Bending over or taking alcohol intensifies the discomfort of an acutely inflamed antrum; temporary reduction of nasal obstruction by a local vasoconstrictor may relieve it Comfort in uncomplicated peptic ulcer is induced by food or an alkali Reduction in mobility of the lower thorax by pressure may allay the pain of acute pleuritis, deep inspiration will increase it The ache of backstrain, arthritis or other musculoskeletal disturbance of the back may decrease when the patient first lies down, become worse on first arising after a rest and temporarily decrease with activity In acute pericarditis, partial relief may be obtained by bending forward or leaning to one side Pain on walking which disappears shortly after activity is stopped *or when a vasodilator is taken is characteristic of angina pectoris*

Certain bodily functions also have a definite effect on pain The influence of eating on peptic ulcer and of exertion on angina pectoris have already been mentioned A bowel movement may temporarily lessen discomfort if the lower bowel is partially obstructed or acutely inflamed, it will increase that of a local lesion in the rectum or anus Chewing or laughing may precipitate an attack of tic douloureux

Not every kind of symptom can be analyzed in exactly this way. A history of some disturbance of bodily function such as poor appetite, loss of weight, or constipation requires a different approach A patient's statement that he has no appetite means little Many perfectly healthy persons go through life dis-

claiming any relish for food. If it can be brought out that there has been a *recent noticeable change in appetite* there is probably mischief afoot, although the cause may not be serious. Nostalgia, grief, unpleasant surroundings or unpalatable food is just as likely a cause as gastric carcinoma or pulmonary tuberculosis. Furthermore, true anorexia may be confused in the patient's mind with reluctance to eat traceable to a sore mouth, ill-fitting denture, pain on swallowing or fear of some actual or imaginary ill effect of eating. Increase in appetite may be due to thyrotoxicosis or diabetes or, on the other hand, to improvement in state of mind or general physical condition.

The fact that a person is overweight or underweight is not necessarily significant. If the weight has been constantly subnormal there is probably nothing seriously wrong, *recent, noteworthy change is important*

Chronic constipation over a period of years is usually not serious. Constipation appearing fairly suddenly in a patient whose bowel habits were previously normal might be the first indication of carcinoma or other obstructive lesion of the gastro-intestinal tract

How much the history of present illness can contribute to the solution of a complex problem is well illustrated by *chronic headache*, often a baffling symptom. The headache of eye strain is apt to occur in the late afternoon as dull, frontal discomfort and may be absent on days when the patient does not work The frontal pain of frontal sinusitis is more acute, more localized, usually follows an acute nasopharyngitis, is worse in the morning when secretions have accumulated overnight, subsides during the day after drainage has been established, but increases again toward evening as fatigue develops Bending low, turning the head suddenly, or drinking alcohol always aggravates it. An acutely infected antrum or sphenoid sinus produces this same type of pain, but in the former the discomfort is localized to the infraorbital region, in the latter, to the occipital region The so-called "nervous" headache of many highstrung or chronically fatigued women is actually a feeling of *tenseness in the back of the neck*, relieved perhaps by a night's sleep, by lying down or by local massage. The discomfort of chronic arthritis of the cervical spine or of chronic neck strain may have similar distribution, but in these cases it is felt most acutely on arising (because of stiffness), subsides during the day, and becomes worse toward evening because of fatigue Usually it can be eased by lying down. *Migraine* headache is entirely different from any of the above. It usually begins in the morning, preceded by some prodromal symptom such as blurring of vision or an empty feeling in the stomach, is characteristically unilateral and may be felt over a small or wide area. It occurs at intervals of days, weeks, or months, with complete freedom between attacks Such phenomena as scotomata, transient restriction of the field of vision or temporary blindness, transient aphasia, or nausea and vomiting accompany it The clinical picture is so typical that it can almost invariably be diagnosed on the basis of history alone (Failure to learn from the history that recurrent attacks of nausea and vomiting have always been accompanied by headache of the migrainous type has in more than one instance led to the unnecessary removal of an appendix or gallbladder.) The headache of brain tumor, usually generalized, is mild at first but rapidly becomes severe

and persistent although there may be short remissions The pain is apt to wake the patient early in the morning and is aggravated by such factors as excitement, exertion or straining which cause cerebral hyperemia.

Past History

Past history begins with an account of where the patient was born and the various places where he has lived A history of residence in the tropics would suggest a group of diseases not to be seriously considered if the patient has always lived in the temperate zone. The general state of health up to the appearance of the present symptoms should be ascertained. Has the patient led a robust, active life, or has he been frail and given to sedentary habits? Certain disorders are more apt to be found in one type than in the other.

A record of previous diseases is next obtained, special attention being given those which might have a bearing on the present illness It is sometimes sufficient to name a malady and obtain an affirmative or negative answer, *when an important clue is being traced, it is advisable to identify the disease for the patient by describing its common symptoms and signs* For example, if rheumatic heart disease is suspected, this method may disclose that what the patient first called "rheumatism" was actually not rheumatic fever, but a mere sprain of the knee or back. Or a patient might state that he once had malaria, the diagnosis having been based on recurrent attacks of chills and fever Close interrogation might elicit other symptoms which would suggest that the chills and fever were due to acute pyelonephritis

Details concerning the course of some previous illness—its severity, duration, and complications—are often helpful

Details of previous operations and severe injuries should be obtained In the case of operations it is sometimes necessary to know exactly what surgical procedure was carried out.

Full information is next obtained regarding the occurrence of previous disorders in particular parts, organs or systems of the body, facts brought out in the present illness naturally indicating emphasis along particular lines Here, again, to name a disorder and accept "yes" or "no" is often insufficient; its common symptoms must be described.

The following order is usually observed.

Head	Neck
Eyes	Cardiorespiratory System
Ears	Gastro-intestinal System
Nose	Genito-urinary System
Tongue	Catamenia
Mouth	Neuromuscular System
Teeth	Weight
Throat	

We continue with chronic headache as an illustration, this time to show the bearing of past history on the present illness The diversity of causes has al-

ready been indicated. All of them must be thoroughly investigated: Has the patient ever had meningitis, encephalitis, syphilis or symptoms suggesting these diseases? Has he ever had trouble with his eyes? Have they ever been examined? Was he given glasses? If he wears them, when were they last tested and changed? Does he have proper light for his work or reading? Has he ever had mastoid trouble or a chronically draining ear? Is he subject to "colds in the head"? Are they of long duration? Has he ever had sinus trouble? Did he ever notice thick, yellow discharge from the nose persisting after a head cold? Have his sinuses been investigated by a nose specialist or been x-rayed? Are his teeth in good condition? Are there any abscessed teeth? Have his third molars erupted or been removed, or are they known to be impacted? Have any other teeth been removed, and have his jaws been x-rayed afterwards to exclude the possibility of retained root-fragments? Has he been subject to stiff neck or difficulty with his back? Has he ever been constipated? For how long? Did the constipation exist for years before the onset of the headache? Has he had any symptoms suggesting lung abscess or bronchiectasis? Has he had hematuria, polyuria, nocturia? Has he in the past been told that he had high blood pressure or kidney trouble? Has he been nervous, had attacks of nervous prostration or breakdown in the past? These are only a few of the questions which may be necessary in an obscure case

Military History

A survey must be made of assignments, performance and illnesses of any patient who served with the armed forces. A history of duty in regions where malaria, filariasis, schistosomiasis and certain other diseases are endemic might be extremely significant The number of hospitalizations and the reasons therefor should be determined Sometimes it may be advisable to obtain a detailed record from the military authorities Repeated hospitalizations for vague complaints, frequent visits to sick call or repeated transfers to different organizations strongly suggest some physical deficiency or inability to adjust to new situations

Equally important, especially from the standpoint of gauging mental status, is the quality of actual performance. Conflicts with authorities, repeated breaches of discipline, and court martials all point toward a psychopathic personality or actual psychosis Unhappily, the same must be said of an unsubstantiated story of many exploits of heroism or numerous high decorations. With all respect to the splendid records of many distinguished soldiers, sailors and airmen, the physician must not lose sight of the fact that dishonorable imposters have been known to pose as heroes

Family History

The health and quality of the family stock are sometimes significant in evaluating a case. Some strains are strong, others weak; some are long-lived, others short-lived. Some families show a definite pattern of emotional instability; others

are stable. Allergic disorders, arteriosclerosis, hypertension, coronary heart disease, diabetes, nephritis, migraine, some nervous and mental disorders and certain types of malformation show a distinct tendency to appear in members of the same family, either in the same or different generations. Syphilis or tuberculosis in a family offers the hazard of contact-infection. The incidence of such diseases as those mentioned above in parents, brothers and sisters and even in grandparents, uncles, aunts and cousins should be ascertained in appropriate cases. The relationship between rubella occurring during the first three months of pregnancy and some congenital defect in the child must be borne in mind. The value of family history is frequently limited by the fact that the patient can give no more definite description of ailments than "change of life", "chronic *indigestion*", or "*complication of diseases*".

Marital History

The health of wife or husband and the children should be determined, with particular reference to acute contagious diseases and to such infections as tuberculosis, syphilis and gonorrhea. In the poorer classes, general ill health of several children suggests dietary deficiencies. The occurrence and number of miscarriages should be noted. Those not induced and occurring in a series uninterrupted by normal births suggest syphilis. The subject of venereal disease as it relates to the patient or his family should be delicately approached.

Occupational History

Much of the daily activity connected with occupation, although customarily treated as a part of occupational history, can be considered to better advantage under *Habits*, when the day-to-day and hour-to-hour life of the patient is being surveyed.

Occupational history should establish the exact nature of the patient's work, with particular emphasis on possible accident and health hazards. Such general terms as "factory worker", "clerk" and "salesman" are of no value. What one wants to know is exactly what the patient does and when he does it. Some common health hazards are: exposure to dust, toxic chemicals or infection, improper ventilation or illumination, abnormal temperatures and accident risks. Plumbers, painters and storage-battery workers, for instance, are in danger of lead poisoning, rubber and patent-leather workers may be exposed to benzol, automobile mechanics, illuminating gas workers and traffic officers, to carbon monoxide.

Habits

Mental and physical fatigue, improper food or eating habits, and the immoderate use of coffee, tea, tobacco, alcohol or drugs are of far more importance in the production of illness than is generally realized. To determine exactly what part any of these factors may be playing in a given case, *we must have a clear and complete picture of the patient's daily routine of living*. His assertion

that he is or is not overworked, that he does or does not eat, smoke or drink in moderation cannot be depended upon. *The physician must form his own opinion on the basis of the actual facts*—not the patient's interpretation of them. He can do so only by obtaining a faithful and minutely detailed account of the patient's eating, sleeping, working, recreational and resting habits.

When does he get up? Is he rushed in the morning? Does he bolt his breakfast and dash for the bus, standing up all the way to work, or does he have a leisurely breakfast and comfortable transportation? Does he allow time in the morning for bowel movement or does he, in his haste, suppress the urge? What is his routine at the office or shop? What physical and mental strain does it involve? Is luncheon an unhurried meal in quiet surroundings or a hastily eaten sandwich in a noisy lunchroom? When he reaches home in the evening, is his dinner already on the table or does he first have a half-hour of relaxation? Does he bring work home, or does he leave business completely behind? Does he have quiet, restful evenings or does a round of dinners or committee meetings or bridge games keep him up late and add to his fatigue? What time does he go to bed? How well does he sleep? Does he have difficulty in falling asleep or does he awaken too early? Are sedatives required and if so, what kind, in what dosage and how often?

Exactly what does he eat and drink at each meal? What constitutes for him a typical breakfast, luncheon, dinner? Does he chew his food or wash it down with fluid? Is the food palatable, well-cooked and eaten in pleasant surroundings? How much and what does he eat or drink between meals? Does he have a mid-morning or mid-afternoon coffee and sandwich or nibble at a candy-bar while he works? Housewives, cooks and others who are in and out of kitchens are apt to nibble at bread or cake or sip innumerable cups of coffee or tea without realizing how much extra food they are taking

How many cups of tea and coffee does he drink? How many cigarettes does he customarily smoke each day? How much liquor does he drink? What medicines does he use and for what purpose? Persons who are conceded to be heavy drinkers cannot, as a rule, be depended upon to give an accurate account of their drinking habits. It may be necessary to go to the family or friends for the facts. Drug addicts are notoriously deceitful and sometimes so secretive that even those close to them may be unaware of the addiction *Many people take large quantities of one or another proprietary preparation, containing alcohol or some habit-forming drug, quite unaware of the dangerous potentialities* Whenever it is necessary to consult family or friends, care must be taken not to create unfair suspicion of the patient or betray his confidence.

What are the patient's avocations? What demands do they make on his time and energy? Does he, after work, play several hard games of squash or tennis and impose added strain on a machine already worn out by the day's work? Does he take vacations? How frequently and for how long? When he is away does he forget business or does he "keep in touch with the office"? No aspect of the patient's life is too trivial. In such minutiae may lie the solution of a complicated diagnostic problem

Social History

Recent trends in medicine and psychiatry have emphasized the influence of environmental situations upon organic disease and the neuroses *Emotional maladjustment often produces symptom complexes which so closely simulate those caused by organic disease that differentiation is extremely difficult* Furthermore, mental state influences physical health by affecting the course of structural disease Excitement is a common precipitant of attacks of angina pectoris; worry is recognized as a frequent cause of reactivation of quiescent peptic ulcer. Dyspnea, precordial pain and palpitation occur in certain types of heart disease, yet any one of them may appear as a result of emotional disturbance in a patient with a perfectly sound heart Chronic indigestion with abdominal discomfort, gas and pain can be induced by psychogenic factors in a person with a normal gastro-intestinal tract and good dietary habits

It is important to evaluate the emotional status of a patient, no matter what the presenting complaint happens to be To do this one must have some conception of his personality traits and his adjustment to life One must know the details of his family and sex problems, his business life and financial worries How far interrogation should go must be decided on the circumstances of the individual case. A well-defined structural case does not necessarily require a detailed social history, an obscure case presenting features not entirely consistent with organic disease or suggesting some emotional factor demands a thorough survey

Moreover, every physician should be able to recognize the psychoneuroses and common psychoses He can often handle the former himself but the diagnosis and management of the psychoses and the more deep-seated neuroses are the province of the psychiatrist Unless one is familiar with the usual patterns of emotional disorders he runs the risk of attributing symptoms to nonexistent structural disease. For example, the diagnosis of valvular heart disease is often incorrectly made in cases of neurocirculatory asthenia Familiarity with the key symptoms of the psychoses may enable one to prevent the suicidal or homicidal attempts so often occurring in the major psychiatric disorders The cardinal

Overcoming the reluctance of the average person to discuss his intimate affairs requires a subtle technique He must be made to feel that he can be frank,

of the patient's needs and submerge his own convictions and prejudices *Surprise or disapproval must never be expressed* Unless the patient can look on the physician as one who is friendly, sympathetic and to be trusted, no approach will succeed in breaking down his reticence on the subject of his intimate life, experiences, fantasies, ambitions and fears.

Almost every patient resents any suggestion of an emotional basis for his

symptoms and receives it with an indignant: "There's nothing wrong with my mind." Promptly he throws up a defense. Questions having to do with his state of mind will elicit evasive replies or none at all With such a person there is little use in inquiring whether anything is troubling him The inevitable reply is: "Nothing". He wants to be told that his symptoms are due to some specific disease. something that can be cured by a pill, a diet or an operation. His ill- *ness must have a label, the more dramatic the better* The term "real" or "imaginary" should therefore be avoided in discussing the symptoms. Furthermore, this concept has no basis in fact and to imply it is not sound medicine.

It is much more satisfactory to have the patient talk about his problems voluntarily but, when necessary, questions may be asked as simply and understandingly as possible. It is best to begin by discussing the more impersonal situations, and as the patient gains confidence in the doctor the more personal and intimate details may be approached *A topic should never be forced:* one at which the patient balks at the first interview, he may be willing to discuss at a later visit. Questions should not be put too baldly; they may be misinterpreted as unnecessary "prying" Facial expression and behavior should be closely observed hesitancy in talking, tendency to change the subject and evidence of upset feelings are all indicators of the emotional value of a topic

The conversation should be guided into such channels as will provide information about the patient's emotional status Is he hypersensitive? Are his feelings easily hurt? Is he quick to tears when crossed or disappointed, or even when reading a sad story or seeing a sad film? Is he happy or depressed? Is he satisfied with his lot in life? Does he have feelings of guilt or unworthiness? Has he been oppressed by unfounded feelings of remorse, suspicion or anxiety? Does a conviction that life has dealt harshly with him make him comment caustically on men who appear successful, blame others for the thwarting of his own ambitions or shun people entirely? Does a sense of inferiority express itself in diffidence and shyness, or lie concealed beneath bluster? Has he had nervous breakdowns? Has he any phobias. fear of crowds, subways, closed rooms? Does he lack friends? Does he attribute this to lack of opportunity or money, or to defects in his own personality? Does he think he is subject to unjust criticism or ridicule for any reason? Is he satisfied with the salary he is receiving? Does he like his position and his work? Does he think his talents are being wasted? Is his job too big for him? Does he get along with the people with whom he works?

Are both work and recreation overshadowed by financial worries? Has he undertaken any business venture which has proved unwise? Is he harassed by debts? Is his time spent inventing excuses to give to creditors? Is the standard of living he has set for himself higher than his income can support? Is he actually finding it difficult to provide his family with the barest necessities? Have medical and hospital bills overwhelmed him?

Questions concerning the patient's life at home must be put with care If the patient is married, are husband and wife in love with each other? Does either suspect the other of dissatisfaction with the marriage? of interest in someone

else? of actual infidelity? What are the grounds for these suspicions? Is there a third person in the picture? Is either one unreasonably jealous of the other? Is she the type of wife who constantly and unfavorably compares her husband with her brother, father or her best friend's husband? Does she embarrass him or deprecate him when they are with friends or relatives? Does she hound him to accompany her to social functions when he is tired and would much prefer an evening at home? Does she fly into tantrums or rages and condemn him for a variety of imagined or real but petty faults? Does she complain about his inability to provide her with the luxuries which her friends are enjoying? Does she run home to her mother with just or unjust complaints? How much is her mother a part of his married life? Has he married a family?

Is the patient's husband a source of fear or distress: domineering, unreasonable, fault-finding, unduly suspicious or jealous, abusive? Does he constantly compare her unfavorably with his mother or his first wife? Do they have common interests or does each seek his or her own entertainment? Does he pay her the little attentions a woman treasures or fancy his obligations are met by a handsome allowance? Does he drink too much? Are there children in the home? Do they fear or dislike him? Are they a source of constant worry because of illness, poor scholarship, restlessness, incorrigibility or maladjustment? Do the parents agree on points of upbringing or is there serious friction, with one constantly setting at naught the disciplinary measures instituted by the other?

Do husband and wife have a circle of friends whose company they both enjoy? Or are they entirely without close friends and left to their own resources? Is either's privacy constantly invaded by visiting relatives?

If the patient is unmarried, how does he feel about his parents, brothers and sisters? Is he domineered by any of them or does he lead his own life? Has he since childhood been afraid of his mother or father? Is his devotion to either unusually profound? Do his parents quarrel? Is he an unwilling witness to their altercations? Is he financially independent or does he rely on one of his parents for support? Is his father forcing him into succeeding to the family business or profession? Does he want instead to be a musician, or writer, an artist? What of love-affairs? Is he in love with someone whom for one reason or another he is unable to marry?

If the patient is a young woman one should find out whether she is popular with young men If not, does she think it is because she lacks opportunity to meet them or because she is unattractive? Is there parental objection to her friends and social activities?

Sex-life is a delicate topic but questions can usually be asked directly without causing offense. If the patient is married, is his sex-life happy? Does he obtain sexual gratification? Does either husband or wife find intimacy repugnant? Is either lacking in libido or actually impotent? Is there a fear of pregnancy? What measures, if any, are being taken to prevent it? Is *coitus interruptus* practiced? Has the husband a mistress or the wife a lover? Does infidelity weigh on the conscience?

Unmarried patients as well as those who are married must often be inter-

rogated concerning sex problems. In some cases questions concerning sexual relations, masturbation, and perversions are necessary, with special effort being made to determine whether their practice is causing anxiety or feelings of guilt.

Questions such as these consume a great deal of time. But without them thorough understanding of a patient and his illness is often impossible The astute physician never loses sight of the fact that one's loves and hates, hopes and fears, ambitions and frustrations can have as real an effect on his health as many disturbances that are detectable by the stethescope, the x-ray or the electrocardiogram.

RECORDING THE HISTORY

The history form given below, modeled on that used in the wards of the Massachusetts General Hospital, is satisfactory for general medical use It observes the customary order for recording the history, which, as already indicated, differs from the order followed in *taking* it.

HISTORY

Name **Residence** ***Age*** ***Sex***

Marital Status **Race** ***Nationality*** ***Occupation***

Chief Complaint:
Nature and duration of presenting symptom

Family History:

Health of father, mother, brothers, sisters, if any are ill, nature of ailment, if deceased, cause of and age at death

Occurrence in present or former generations of malignant disease, diabetes, gout, tuberculosis, arthritis, diseases of hematopoietic, circulatory, respiratory, digestive, renal, nervous, or muscular systems

Past History:

Birthplace.

Former places of residence: approximate time in each

General health up to onset of present illness: Immunizations Exposure to tuberculosis and other infections

Previous illnesses. Including measles, rubella, mumps, chicken-pox, pertussis, influenza, scarlet fever, diphtheria, typhoid fever, bronchitis, asthma, pneumonia, pleurisy, tonsillitis, rheumatism or rheumatic fever, chorea, smallpox, tuberculosis, malaria, or any other severe disorders. Details of any of these illnesses
Exposure to tuberculosis

Operations: Date of each operation, nature, anesthetic used, complications, and length of time patient incapacitated.

Injuries: Details concerning any severe injuries

Head: Headache, vertigo.

Eyes: Date of last examination, glasses; vision, inflammation, pain

Ears: Hearing, tinnitus; pain, discharge

Nose: Head colds, discharge, epistaxis, obstruction, sense of smell

Teeth. General condition; previous x-rays, abscesses, impacted molars or retained root fragments; extractions; dentures

Tongue: Sense of taste, paresthesias, pain or soreness

Mouth: Pain, local lesions, salivation; sore or bleeding gums

Throat: Sore throat; tonsillitis, peritonsillar abscess, hoarseness; speech defect, difficulty in swallowing

Neck: Stiffness, pain; swellings, pulsations, venous distention.

Cardiorespiratory: Pain in chest; palpitation; dyspnea, orthopnea; cough; sputum, amount and character, hemoptysis; night sweats

Gastro-intestinal: Appetite; bowels, cathartics, nausea; gas; vomiting, hematemesis, distress, pain; colic; icterus, diarrhea, bloody, tarry, or clay colored stools; hemorrhoids.

Genito-urinary: Usual number of day and night voidings, recent change, frequency; nocturia, retention; incontinence, dysuria, bloody, smoky, cloudy, or purulent urine, *penile discharge History of syphilis—primary lesion, lymphnodes, mucous* patches, falling hair, rash.

Catamenia: Age at onset, frequency, regularity, duration, pain, amount, leukorrhea; date of last period, any recent change in cycle, date of menopause, menopausal symptoms, bleeding since menopause

Neuromuscular: Fainting or other forms of unconsciousness twitching fibrillation, spasm, or convulsions girdle, shooting, muscular, or joint pains, anesthesia or paresthesia; stiffness, restriction of motion, weakness, incoordination of movements, or paralysis

Skin Changes in color, texture, or character, eruptions or other lesions

Weight. Amount and date of greatest weight Loss or gain of weight, over how long a period Any known cause.

Military History:

Approximate dates of enlistment and discharge, type of discharge

Stations and theaters of duty, military occupational specialty and nature of duty *performed*

Immunizations· illnesses, reports at sick call and hospitalizations with reasons therefor

Relationships to companions, superiors, subordinates, conflicts with military authorities

Decorations, battle awards

Occupational History:

Place of employment and exact nature of the work in which the patient is engaged (*e.g.*, iron foundry, moulder's helper) Length of employment in the industry and in the particular position now occupied If employment has been varied or irregular, nature of previous work Where indicated, existence of industrial health hazards, such as dust, toxic agents, poor illumination and ventilation

Marital History:

How long married

Health of husband or wife, if ill, nature of ailment, if deceased, cause of death

Number of children and state of their health, if any are ill, nature of ailment, if deceased, cause of death

Miscarriages Causes if known, chronologic relationship to normal births

Habits:

Daily routine, including time of arising and retiring, hours of work, eating habits, typical meals, use of tea, coffee, tobacco, alcohol, and drugs, avocations, recreation, vacations

Social History:

Patient's evaluation of temperament, memory, judgment, mental ability Physician's impression of patient's personality, emotional make-up, reliability, particularly with respect to the facts given in the history Facts concerning his family, business, and personal life which might have a bearing on present illness

Present Illness:

An orderly and, as far as possible, chronologic account of the development of the patient's illness from the appearance of the first symptom to the present time Detailed description of each symptom and its relationship to other symptoms and to normal bodily functions Degree to which patient has been incapacitated Which symptoms have caused the incapacity? Previous treatment and its effects

2

PHYSICAL EXAMINATION

GENERAL CONSIDERATIONS

The four basic procedures used in conducting a general physical examination are indicated below. These, as well as various special methods used, such as testing for impaired sensation or motor function, are described in subsequent chapters.

Inspection. Looking at the body

Palpation. Feeling the various parts

Percussion. Listening to the sounds produced and noting the degree of resistance encountered when a region is tapped with the fingers or a percussion instrument.

Auscultation. Listening to the sounds produced within the body by physiologic or pathologic processes

Actual examination by these methods is performed after the history has been taken. But the physician whose perception has been sharpened by experience consciously or unconsciously begins his examination as soon as he sees the patient. From the general appearance and facial expression may be gathered the impression of agitation, worry, depression, exhaustion, strain, good or bad health, pain or distress Obesity or emaciation, many gross deformities, unusual appearance of the skin, abnormal carriage, gait, or position in bed are noted at a glance.

While the history is being taken, personality traits, unusual mannerisms and the character of the voice and speech are revealed Mental alertness or dulness, a voice which breaks with nervousness or anxiety, eyes that shift furtively, or a manner expressive of emotional instability often provide valuable clues

At the bedside, the first glance will often suffice to decide whether the patient is or is not seriously ill Experience teaches one to recognize the general clinical pictures of certain illnesses. the anxious, distressed expression and jerky grunting respirations of pneumonia, the labored breathing and upright position of congestive heart failure, the mental alertness, shining eyes and flushed face of severe sepsis, the grayish cyanosis and the sweating of severe circulatory collapse.

Many disorders—thyrotoxicosis, chorea, paralysis agitans, tabes dorsalis, advanced pernicious anemia—to name but a few, are often suggested by the

first impression. *"Snap diagnosis" is never dependable;* it must always be confirmed by further investigation.

Generally speaking, every patient should be given a thorough physical examination. Naturally those parts of the body to which history has directed suspicion merit the most detailed investigation, but not to the exclusion of others *Important disturbances frequently exist without producing subjective manifestations and are often found incidentally on physical examination.* Just how detailed physical examination should be depends on the nature of the individual case. The obscure one demands an exhaustive examination, in the obvious one, some of the details can often be dispensed with. In pneumonia, for example, the more detailed neurologic tests would hardly be necessary, yet the cardinal signs of a complicating meningitis should be looked for. Nor, in a female, would a vaginal examination be necessary unless there were reason to suspect that the pulmonic lesion might be an embolic process secondary to uterine infection from puerperal sepsis or some other cause

Sometimes the condition of the patient is such that any disturbing or prolonged examination might have disastrous results Only the measures necessary for meeting the immediate emergency should be *carried out* *Such would be* the case in a patient with recent pulmonary or gastro-intestinal hemorrhage, acute myocardial infarction or large pulmonic embolism

In all but the briefest illnesses, a single examination is rarely sufficient. *Re-examination from time to time is essential in order to follow the progress of* a case and discover complications if and when they develop Again one must beware of overzealousness To keep well posted, it is often sufficient to merely look at the patient, study his chart, feel his pulse, and briefly examine certain parts.

Physical examination must be conducted in a warm, well-lighted, quiet room Daylight is preferable because such color variations as pallor, jaundice and moderate cyanosis, certain skin eruptions and other abnormalities can be easily overlooked in artificial light The patient *should disrobe completely.* But only the region being examined need be exposed, a sheet or loose garment covering the rest of the body His comfort demands a warm room

The physician should train himself to follow an identical routine in every case, passing from one part to another in a particular order An important detail may be inadvertently omitted by skipping about haphazardly.

RECORDING THE PHYSICAL EXAMINATION

The form presented below based on that in use at the Massachusetts General Hospital is recommended as a guide for general physical examination. Intended only as a check list, it should be supplemented by a detailed description of all pertinent findings Modifications to suit the individual case are permissible. The indicated order of examination need not be rigidly adhered to After such facts as general appearance, temperature, *et cetera* have been noted, it is often more convenient for patient and physician if the back, and the posterior aspects of the head, neck, and lungs are examined first Then,

starting with the anterior aspect of the head, one can proceed downward in the usual order. Vaginal and rectal examinations are left until last. They may be postponed or omitted but are too often neglected, and *serious mistakes often result from failure to investigate these regions.* Detailed orthopedic and neurologic examinations, if necessary, can be more conveniently carried out after the more routine procedures have been completed.

The diagrams at the end of the form are useful for indicating the sites of abnormal findings.

PHYSICAL EXAMINATION

Name **Date** , .

Temperature **Pulse Rate** **Respiratory Rate** **Height** **Weight**

General Appearance: Physical Development Nutrition
Stature Posture Gait
Tremors or Other Abnormal Movements

Mental Status: Degree of Consciousness Cooperation
Orientation Emotional State

Skin: Color Texture Moisture
Eruption Other Lesions

Head: Appearance Size Shape
Hair—Distribution Character
Scalp

Face: Appearance Color Tenderness
Abnormal Movements

Eyes: *Brows* *Lids* *Vision*
Eyeballs—Motion Prominence Tension
Conjunctivae Sclerae Corneae Irises
Pupils—Size Shape Regularity Reactions
Fields Fundi

Ears: Appearance Hearing
Tenderness Discharge
Drums Canals
Postauricular Swelling or Tenderness

Nose: Appearance Discharge
Nasal Cavity Septum
Transillumination Smell

Mouth: Breath
Lips—Color Fissures Lesions
Tongue—Tremor Deviation
Color Moisture Texture
Gingivae—Color Texture Recession Bleeding
Lesions Metal Line Pus

	Teeth—Number Condition Dentures Buccal Mucosa—Color Eruptions Lesions
Throat:	Palate and Uvula Pillars Tonsils Posterior Pharynx Larynx
Neck:	Lymphnodes Scars Thyroid Salivary Glands Position Motion Veins Pulsation Edema Trachea
Shoulder Girdle:	Deformity Swelling Tenderness Freedom of Movements Muscle Spasm Supraclavicular or Infraclavicular Lymphnodes
Arms and Hands:	Position Deformities Color Tenderness Muscle Strength Abnormal Motions Joints Edema Freedom of Movements Temperature Moisture Local Swellings Vessels Epitrochlear Lymphnodes Fingers Nails Reflexes
Breasts:	Size Contour Tenderness Masses Glandular Consistency Nipples Skin—Color Texture Retraction
Axillae:	Skin Hair Lymphnodes
Back:	Deformities Freedom of Movements Skin Swelling Tenderness Muscle Spasm Scapulae
Thorax:	Shape Respiratory Movements Swelling Pulsation
Heart:	Apex Impulse—Location Character Thrill Measurements—Right Border Left Border Supracardiac Dulness Midclavicular Line Sounds—Rate Rhythm Intensity Quality Murmurs Pulses—Equality Character Condition of *Temporal, Brachial, Radial Vessels* Blood Pressure
Lungs:	Resonance Diaphragmatic Excursion *Tactile Fremitus* Breath Sounds Whispered Voice Sounds Spoken Voice Sounds Rales

Abdomen: Size Shape Scars Tension Percussion Note
Masses . Tenderness Spasm
Pulsation . Umbilicus .
Fluid Wave . Shifting Du'ness .
Liver—Dulness Edge .
Spleen Kidneys .
Costovertebral Tenderness Herniae . .
Peristalsis—Audible . Visible ..
Bladder—Dulness Palpable Distention
Inguinal Lymphnodes . Reflexes . .

Legs and Feet: Position Deformities Color Tenderness ..
Muscle Strength .
Abnormal Motions Freedom of Movements
Joints Edema
Temperature Moisture .
Local Swellings Ulcerations .
Vessels Shins Arches
Toes Nails
Reflexes Clonus Kernig Romberg

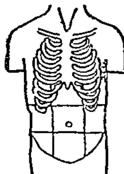
Male Genitalia: Pubic Hair Penis Scrotum
Testicles Epididymis Cords

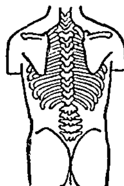
Female Genitalia: Pubic Hair Vulva Vagina
Perineum Cervix Fundus Vaults

Rectum: Anal Orifice Sphincter Tone Fissures
Hemorrhoids Tenderness
Internal—Obstruction Masses Prostate
Tenderness

Other Abnormal Findings:

Recapitulation of Important Variants: .





3

THE BODY AS A WHOLE

STATURE

Dwarfism

This term is applied to persons who, according to the norm for their race, are unusually small Some dwarfs are normally proportioned and are normal sexually. The variant may be due to a familial tendency to small stature in spite of normal pituitary function In some cases specific deficiency of the anterior pituitary growth hormone has been suspected but never proved

Pituitary Dwarfism. *Growth as a whole is curtailed The head and trunk* are normally shaped but small Because of failure of their epiphyses to close at the usual time, the extremities are disproportionately long. The figure is delicately formed, resembling that of a child The features assume early the appearance of age (*progeria*) Development of genital organs and secondary sexual characteristics is retarded

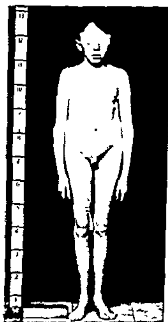


Fig. 3 1. Hypopituitarism Age 18 Small stature with absence of primary and secondary sexual development *Progeria present but lost in reproduction*

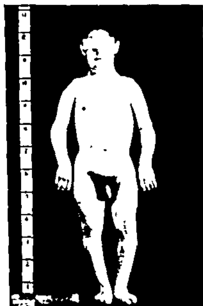


FIG 3 2 Achondroplastic dwarf Age 17

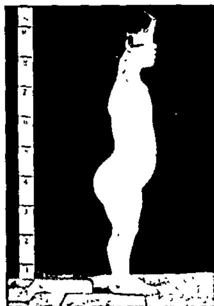


FIG 3.3 Achondroplastic dwarf Age 8

Achondroplasia. The head and trunk approach adult size but the extremities, because of failure to develop normally, are short and bowed with resultant striking disparity between the size of head and trunk and the length of the limbs. The back shows extreme lumbar lordosis In the male the genitals are relatively large.

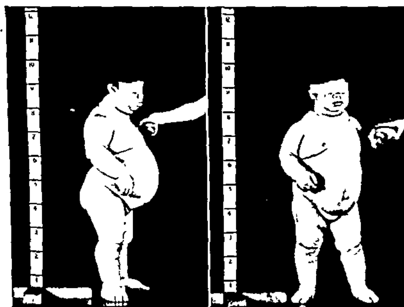


FIG 3 4. Cretin Age 24.

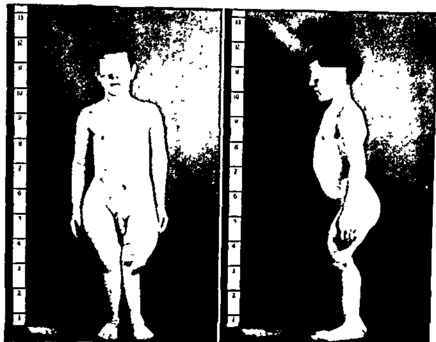


FIG 3 5 Rachitic dwarf Age 14

Cretinism. In contrast to achondroplasia, growth of all parts is retarded so that the bodily proportions of infancy are retained Outstanding features are a relatively large head, thick lips and eyelids, large and protruding tongue, short and thick trunk, dry, yellowish skin, pot-belly with umbilical hernia, spade-like hands and feet, and perhaps deformed legs. The infant has a characteristic croaking cry, when the speaking voice develops it too has a hoarse or croaking quality

Dwarfism can also be brought about by rickets and other deficiency states in childhood as a result of improper development and deformity of the long bones and back Any chronic disease and even malnutrition alone occurring during the growth period may result in a small stature and poor development Deformity of the spine, as in spinal tuberculosis with kyphosis, may be either the sole or contributing cause of short stature.

GIANTISM

Many persons are unusually tall by virtue of an inherited tendency. Otherwise abnormally large stature is usually the result of excessive production of the anterior pituitary growth hormone beginning *before* onset of puberty. Tumor is the most common cause. In these patients, as in the normal person, there is little if any production of gonadal hormones during the prepubertal period. Gonadal hormones are necessary for normal epiphyseal closure. Consequently during the prepubertal period the excessive amount of growth hormone causes abnormal linear growth of the long bones and the body becomes abnormally tall. If tumor destroys most of the remaining normal pituitary

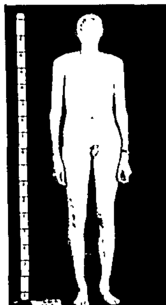


FIG 3 6 Eunuchoid build. Age 16 Disproportionately long extremities, small genitalia, and absence of secondary sex characteristics

gland and hence impairs production of gonadotropic hormone, puberty is delayed or absent with resultant retardation or absence of sexual development. If the gonadal hormones do appear and the epiphyses close at puberty, overproduction of growth hormone then results in acromegaly.

Acromegaly. The cause is overproduction of the anterior pituitary growth hormone *after* closure of the epiphyses The head is large, the supraorbital



FIG 3 7 Acromegaly

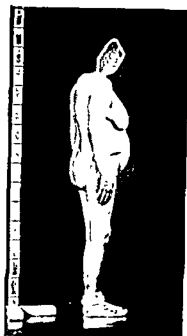


Fig 3 10 Cushing's syndrome Age 24 Abdomen protruded Extremities not enlarged Abdominal striae present but lost in reproduction

the hips wide Knock-knees are common; they often become less pronounced with weight reduction and sexual maturity Sex organs are underdeveloped and secondary sex characteristics absent. In the male the testes may be undescended. Metabolism may be low and sugar tolerance high Many of these children develop normally without treatment or with dietary regulation Others remain sexually underdeveloped, their obesity may or may not respond to dietary measures

The term *Froehlich's syndrome* is often incorrectly applied to this group Actually the case originally described by Froehlich was one of obesity, retarded growth and hypogenitalism associated with tumor in the pituitary region The syndrome is exceedingly rare.

Cushing's Syndrome. The manifestations are due to overactivity of the adrenal cortex resulting from tumor or hyperplasia Whether or not the latter can result from basophilic adenoma of the pituitary is not clear However, improvement of many of these cases following irradiation of the pituitary suggests a primary disorder of this gland The syndrome is much more common in females The trunk appears fat, the limbs remain relatively normal The obese appearance is due in part to pot-belly resulting from weakness of the abdominal muscles and to shortening of the spine secondary to osteoporosis Thinning of the skin with resultant prominence of its blood vessels gives the patient a plethoric appearance and contributes to easy bruising Other characteristics are general muscular weakness, purple abdominal striae, hypertension, insulin-resistant diabetes, and, in women, amenorrhea and hirsutism without other evidence of masculinization Males are apt to become impotent.



Fig 3 11 Adiposis dolorosa

Adiposis Dolorosa. Fat appears subcutaneously in irregular masses, which are painful and tender This is also known as *Dercum's disease*

POSTURE AND POSITION

The healthy, well-developed body has an erect carriage, straight shoulders, a full chest, flat abdomen and firm muscles The asthenic person is apt to have a poorly developed figure with exaggeration of the normal spinal curves, drooping shoulders, flat or sunken chest, narrow costal angle, prominent abdomen and poor muscular development Such a figure does not necessarily denote disease. It is often seen in healthy persons as well as in chronic debilitating illnesses

A patient is often impelled by his ailment to hold himself in a peculiar position while standing, sitting, or lying in bed A stiff neck causes the head to be held at an odd angle. The patient with a lame back will assume an unusual attitude, chosen for the relief it affords from the strain on the affected muscles or ligaments With severe dyspnea, the patient sits upright, sometimes bent






Fig 3 12 Posture standards (Courtesy Children's Bureau, U S Dept of Labor)

slightly forward The pain of acute pleuritis or acute pericarditis may be partially relieved by lying on one side, bending forward or holding the body in some abnormal position A patient with severe mediastinal disease is likely to sit bent forward even when asleep One with an acute intra-abdominal disorder may prefer to lie on his back or side with the legs drawn up to relieve tension on the irritated abdominal muscles; in colic the trunk may be bent forward and the thighs pressed against the abdomen In severe meningitis or tetanus, the patient lies with the body rigid, the back arched and the head in extreme extension (*opisthotonos*) A patient with an inflamed joint usually holds the limb partially flexed

GROSS DEFORMITIES

The trained observer will note at a glance such deviations as harelip, paralytic facies, the flail arm of old infantile paralysis, the adducted, flexed arm, hand and fingers of hemiplegia, the high, stooping shoulders and increased anteroposterior diameter of the thorax associated with emphysema or chronic asthma, spinal deformities such as marked kyphosis, lordosis and scoliosis, atrophy or abnormal position of the leg in such disorders as tuberculous disease of the

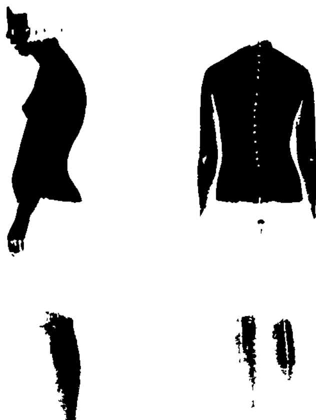


Fig 3 13 Poor posture Mild scoliosis also evident in posterior view.

hip, infantile paralysis and hemiplegia, and bowing as in rickets or Paget's disease.

GAIT

An important clue to diagnosis is often provided by the manner of walking. In tabes dorsalis the patient is ataxic, walks on a wide base and keeps his eyes focused on the ground; his legs are lifted high and the feet flop to the ground. In hemiplegia the affected leg is held stiffly; it swings outward and the foot is rotated inward as the patient walks The paraplegic's legs are stiff and in walking he shows a tendency to shuffle and to cross the legs In multiple sclerosis there are spasticity and staggering The gait of cerebellar disease is staggering, there is also lack of coordination between movements of the trunk and arms and the action of the lower extremities Attempts to walk in a straight line result in veering to one side In the Parkinsonian syndrome, the neck, body and limbs are stiff and the trunk is bent foreward, to one side or, rarely, backward. Steps are

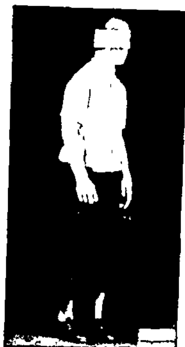


FIG 3 14 Bilateral birth injury with spastic quadriplegia. Patient showed spasticity and impairment of associated movements



FIG 3 15 Spastic paraplegia showing tendency to scissors gait

short, the gait shuffling and a fall seems imminent As the patient with advanced disease walks along, he involuntarily accelerates to a fast shuffling trot (*propulsion or festination*) in order to maintain balance. In hysteria one sees a variety of gaits; some are difficult to differentiate from those of neurologic



FIG 3 16 Typical posture in Parkinson's syndrome

disease, while others are bizarre and do not conform to the more familiar patterns. Severe back strain or disease of the spine causes one to hold himself stiffly. To avoid jarring or pull on the affected muscles or ligaments, he turns his body, sits down, gets in or out of bed, or otherwise changes position with extraordinary care His face is expressive of the effort or discomfort associated with these movements Because of the extra weight in the abdomen, pregnancy, ascites or a large abdominal tumor affects normal balance, to maintain equilibrium the person walks in an ungainly, straddling fashion, with shoulders thrust back, abdomen forward and legs wide apart A local disorder of the pelvic girdle or a lower extremity may cause limping

ABNORMAL MOVEMENTS

Restless Movements. These are encountered in a variety of disturbances, among the most common of which are: pain or distress, mental agitation, thyrotoxicosis, chorea, and alcohol or drug addiction, especially when the habitual dosage is reduced or witheld. Restlessness in bed can occur in almost every serious illness, it is often a bad sign

Choreiform Movements. Spasmodic, purposeless, reiterated gestures of some part or parts of the body are observed The face grimaces repeatedly, or one or more extremities, usually the upper, toss and jerk about in irregular flexion and extension, abduction and adduction In severe cases, almost all the muscles are affected and the movements are very active In their mildest form choreiform movements may be mistaken for mere restlessness, a history of fairly sudden onset or offset points toward Sydenham's chorea

Tremor. Fine or coarse, rapid or slow, rhythmic oscillations affect the head, eyelids, lips, jaw, tongue or extremities Depending on the cause, they may be

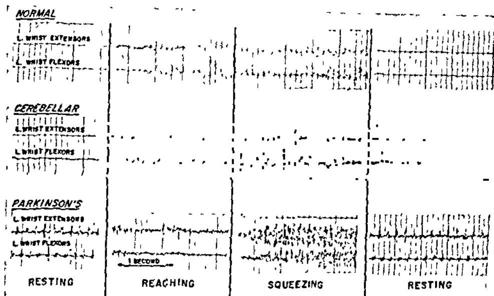


FIG 3 17 Electromyograms (inkwriting oscillograph) with surface electrodes over extensor and flexor muscles controlling movements of fingers and wrist *Resting* Normal No activity Cerebellar disease No activity Parkinson's syndrome A characteristic to and fro tremor created by bursts due to alternating flexor and extensor discharges *Reaching for rubber bulb on nearby table* Normal A variety of electrical patterns but no tremor Cerebellar disease Marked regular tremor, 5 per sec, (intention tremor) Parkinson's syndrome Tremor disappears as a result of activity *Squeezing bulb* A steady discharge in all three subjects *Resting* Return to original status with tremor again evident in Parkinson's syndrome (Courtesy Dr Robert S Schwab)

diminished by resting the part and increased by using it, or *vice versa* Tremor is common in some families, in old age, states of excitement and temporary extreme fatigue. It is also seen in thyrotoxicosis, various toxic disturbances such as alcoholism and chemical poisoning, multiple sclerosis, Parkinsonian syndrome, other neurologic disorders and hysteria Tremor of the hands is further discussed in chapter 6

Tic. This term refers to repeated habit spasms of a muscle or group of muscles such as the constant opening and shutting of the eyelids *(blepharospasm)* Repeated sudden jerking of the head to one side and repeated twitching of the muscles of one side of the face are two other common forms of tic

Convulsion. A seizure characterized by violent, involuntary muscular contractions either generalized or limited to one or more groups of muscles is called a convulsion The contractions may be tonic or clonic. Such seizures occur in uremia, eclampsia, apoplexy, tetanus, tetany, hypoglycemia, hysteria, various neurologic disorders such as meningitis, intracranial tumor, and from unknown cause In infants and young children convulsions are not uncommon during an infectious disease or some minor ailment; here they do not ordinarily indicate serious trouble.

Athetosis. This term refers to repeated irregular, twisting movements, usually of the upper extremities, particularly the hands and fingers. It may be seen

following encephalitis, cerebral vascular accident, and in certain other neurologic disturbances.

SPEECH

Cultural background, emotional make up and sometimes traits of personality or character are reflected by a person's voice and manner of speaking. Nervousness is betrayed by a high-pitched, breathless, uneven voice; agitation by a broken voice; calmness and poise by even, well modulated tones The domineering, insensitive type of individual may bluster; this manner may however, mask an inner feeling of inferiority or insecurity In the shy or oversensitive, the voice may be muffled and barely audible Ordinarily, deaf mutes cannot talk because of inability to learn by hearing but by special training many can be taught to make themselves understood verbally.

In general, articulate speech may be impaired or lost in three ways, although often a combination of these factors is operative. They are

Dysarthria. Speech is indistinct because of defects in the peripheral organs of articulation. Common causes are absence of teeth, ill-fitting dentures, a local disturbance in the mouth or nose, such as severe stomatitis, peritonsillar abscess, harelip, cleft palate, nasal obstruction, sinusitis, or inability to open the mouth properly, as in trismus secondary to an alveolar abscess around a posterior tooth Acute nasopharyngitis, adenoids and sinusitis cause a nasal voice In mild diphtheritic or bulbar paralysis the voice is also nasal, in severe cases it is decidedly indistinct because of lack of muscular control Hoarseness or aphonia occurs in disease of the larynx or interference with its nerve supply.

Seriously ill or debilitated patients may lack the strength to speak audibly. In myasthenia gravis the voice is weak and may show minor defects at the beginning of conversation, it becomes weaker and less intelligible as conversation continues Eventually increasing muscular fatigue may entirely prevent talking but the power returns after a few moment's rest. Following cerebral hemorrhage, thrombosis or embolism the voice may be thick and the words enunciated with difficulty, in general paresis the words are slurred and trouble is experienced in enunciating certain test phrases A dreary monotone without inflection characterizes Parkinsonian syndrome In chorea, speech is jerky, sometimes hesitating, in multiple sclerosis and Friedreich's ataxia it is often scanning or explosive Slurring or thickness of speech is common in alcoholic or drug intoxication The myxedematous patient speaks slowly and monotonously with a hoarse, low-pitched tone

Impaired General Psychologic Function. Here we refer to a generalized disturbance of the intellectual processes In congenital mental retardation, depending on its severity, the patient may not speak at all, may utter unintelligible jargon or show lesser variations which are sufficient to indicate intellectual underdevelopment In such cases, talking is often accompanied by grimacing From this group must be excluded the rare case of congenital word deafness in which the patient is neither deaf nor mentally retarded but has a remedial disorder related to inability to understand spoken language The depressed

person speaks slowly, softly or not at all; if questioned he will either not respond or do so in monosyllables. In maniacal states speech is likely to be loud, rapid and show flight of ideas, often grandiose. A normally genteel person may use a plethora of obscene and profane words In hysteria the patient may whisper or be totally speechless (*hysterical aphonia*) without any evidence of laryngeal disease *Mumbling, thick, unintelligible or senseless speech is encountered in* patients who are stuporous from any cause

Aphasia. Speech is impaired by a local structural defect in a specialized area of the brain which is indispensable for language function In pure motor aphasia other processes such as thinking and remembering may be unimpaired. The patient may, by expression and gestures, make it clear that he understands what he hears or reads but attempts to express himself verbally do not result in clear speech.

Common causes of aphasia are cerebral vascular accident, tumor, abscess or trauma, less often syphilis and encephalitis Pure aphasia is rare. In many cases brain damage is such that the speaking difficulty represents a combination of aphasia, dysarthria and impaired intellectual processes. Transient aphasia may be encountered in cerebral vascular disease, general paresis, an attack of migraine, uremia and following an epileptiform seizure Aphasia is further discussed in chapter 36

THE SKIN

Because significant changes, especially those of color, can be overlooked in artificial light, the skin should always be examined in daylight. In evaluating the significance of skin color and texture, such factors as racial differences, degree of exposure to the elements, occupation and age must be taken into consideration In a doubtful case, one may have to rely on the statement of the patient or his relatives that the color or texture of his skin has changed One must also look and feel for the scars of previous diseases, injuries, or operations, and for alterations brought about by systemic disorders or by disturbances generally considered the province of the dermatologist. The purely dermatologic disorders and the rarer systemic diseases are not included in the following discussion.

Changes in Color

Albinism. Congenital lack of pigmentation may be patchy or all of the skin may be unusually white or pinkish.

Vitiligo. Here one sees various-sized circumscribed patches which lack pigment; each is surrounded by increased pigmentation. The contrast is more apparent following exposure to the sun.

Increased Pigmentation. This may be general or local and due to systemic or local cause. The change is usually due to increased deposits of melanin, carotin, hemosiderin or some metallic pigment.

Generalized Pigmentation This is usually most pronounced in the folds of the skin, on exposed areas, or where there is pressure from clothing. The dis-

coloration tends to be yellowish or brownish. Among the causes of yellowish pigmentation are myxedema, severe pernicious anemia, diabetes, carotinemia, jaundice and prolonged use of quinacrine, dinitrophenol or certain other yellow chemicals. Discoloration varying from a brownish-yellow to a deep brown, depending largely on the severity and duration of the disease, is seen in a variety of disturbances, among the chief of which are Addison's disease, hemochromatosis, chronic malaria and acromegaly. It is sometimes present in association with cachexia in the late stages of hepatic cirrhosis, carcinomatosis, lymphoblastoma and other forms of malignant disease. In thyrotoxicosis, one occasionally sees pigmentation not unlike that of Addison's disease, or patchy pigmentation surrounding areas of vitiligo In pellagra one finds increased pigmentation of the exposed parts, especially the backs of the hands and forearms, often the face and neck, and also over bony points and areas of clothing friction. Exposure to sunlight enhances the change In vitamin A deficiency, diffuse darkening may occur, as well as deeper pigmentation of the hyperkeratotic lesions. Patchy *or diffuse pigmentation is also seen in vitamin C deficiency and sprue Following* prolonged use of arsenic, diffuse or sometimes flecked brown discoloration may occur, often simulating that seen in Addison's disease; keratosis of palms and soles is an important clue. Argyria is characterized by a bluish- or slate-gray color due to deposits of silver salts in the tissues; rarely a similar picture is seen following prolonged use of bismuth

Localized Pigmentation This is seen following repeated irritation, as by constant scratching, frequent application of heat or counter-irritants, and after radiation therapy In rheumatoid arthritis, patchy or diffuse areas of darkening often appear on the extremities, or sometimes the face In older persons, especially women at the time of menopause, various-sized, brownish, flat, noninflammatory spots are likely to appear on the face, hands and occasionally elsewhere (*chloasma*). During pregnancy and in some ovarian disorders similar patches may appear on the face, especially the cheeks, forehead and upper extremities; the mammary areolae and the linea alba darken When the pigmentation is due to pregnancy, fading usually occurs during the subsequent few months In longstanding cirrhosis of the liver, one may observe dirty, grayish-black pigmentation on the dorsums of the hands and forearms and exposed surfaces of the neck This discoloration, presumably due to melanin deposits, tends to increase as the disease progresses Patchy pigmentation is also found in the active or healed lesions of various dermatologic disorders. Brownish discoloration of the lower legs is a common result of chronic impairment of venous flow by thrombophlebitis or varicosities. Small deep brown- to blue-black spots are found in drug addicts on the extremities and trunk at the sites of previous hypodermic injections

Redness Transient redness is often due to overheating or extreme cold. It is common in moments of stress or emotion Redness is seen in areas of inflammation, such as burns or local infection, in many cases of fever, in the exanthematous diseases, especially scarlet fever, and with many other skin eruptions Sunburn is a common cause

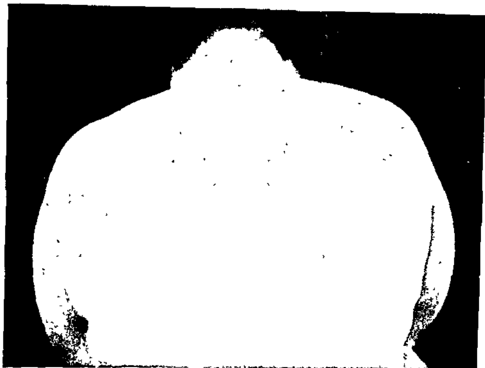


Fig 3 18 Dermatographia

Dermatographia. This is a color change frequently noted during the course of a routine examination If the skin is mildly irritated, as by lightly stroking it with a fingernail, pencil or other sharp instrument, a red, slightly elevated line will soon appear and last for three or four minutes. Following this, the color disappears from the center, leaving a pallid or white line bordered with red Such a lesion—actually a linear wheal—is evidence of vasomotor instability and is most common in urticaria.

Jaundice. Cyanosis. Pallor. These are often more noticeable on the face than elsewhere and are described in Chapter 4 Cyanosis or pallor resulting from local vascular disturbance may be limited to a part or all of one or more extremities

Other Changes

Looseness. A wrinkled, loose skin is seen in old persons as a result of loss of elasticity, with or without loss of weight In others, it is an indication of recent loss of flesh or body fluid.

Tenseness. This is found where there is edema, inflammation or swelling of the tissues beneath the skin from other cause such as hematoma or tumor

Dryness. Abnormal dryness of the skin is found in old age, dehydration from any cause, myxedema, deficiency of vitamin A, scurvy, ichthyosis, scleroderma, diabetes, chronic nephritis, and following large doses of belladonna or atropine

Moistness. Excessive moisture is associated with overheating, excitement, fever, thyrotoxicosis and emotional instability. A cold clammy skin is a serious sign in peripheral circulatory failure.

Atrophy. Because of diminished thickness of one or more layers, the skin is shiny, has lost its elasticity and feels thin, or in extreme instances, like tissue paper. Hair follicles and sebaceous and sweat glands may be destroyed.

Generalized Atrophy. Generalized atrophy of mild or moderate degree is common in old age

Localized Atrophy. This occurs as a result of pressure, nutritional, neurotrophic or circulatory disturbances, as a sequela of various dermatoses, and following intensive radiation. It is common around arthritic joints.

Striae. Pinkish-white or grayish lines of atrophy surrounded by normal skin appear on any part of the body where the tissues have been overstretched They vary in length, usually 1–4 cm. They are most often seen on the abdomen and breasts in women who are or have been pregnant. They are also common on the abdominal wall when it has been stretched by some cause such as obesity, tumor or ascites, and on the extremities in obesity. In many normal women, striae occur on the thighs probably as a result of their enlargement at puberty.

Hypertrophy. There is an increase in the number or size of the cells in one or more layers of the skin, most commonly the epidermal.

Generalized Hypertrophy. This is seen in acromegaly and myxedema In the latter, infiltration by a mucin-like substance causes a swollen appearance but in contrast to true edema there is no pitting on pressure. Vitamin A deficiency is characterized by dry, rough skin, the roughness being due to horny, conical papules at the sites of hair follicles; areas of predilection are the thighs, arms and buttocks Vitamin C deficiency produces similar changes, with the added feature that in the advanced stages the bleeding tendency causes minute hemorrhages about the follicles

Localized Hypertrophy This is seen congenitally (*moles*), and in old age (*senile keratoses*) It also occurs from mechanical irritation as in calluses or corns, infection as in the lesions of blastomycosis and the hyperkeratotic changes of epidermophytosis, and toxic agents such as arsenic In acne rosacea all the layers of the affected region are hypertrophied, in the advanced case the nose may become bulbous, red and enlarged (*rhinophyma*)

Spider Angiomas. Seen in cirrhosis and sometimes acute parenchymal disease of the liver, these are described in chapter 31

Xanthoma In certain cases of disordered lipid metabolism, circumscribed collections of lipid material may be deposited in the skin, tendon sheaths, arterial walls, lymphnodes, or rarely, other structures Those which appear on the surface vary in size from 1 mm to 1–2 cm, are chamois to reddish-yellow, usually soft and either flat or raised The overlying epidermis remains intact Adjacent lesions tend to coalesce

Xanthoma is most often seen on the eyelids (*xanthoma palpebrarum*) The initial lesion begins as a small spot usually but not necessarily near the inner canthus It gradually enlarges and in time others appear and grow. Bilateral involvement is the rule Coalescence is common, regression possible. This form is frequent without known cause, especially in women beyond middle age It is also encountered in hereditary hypercholesterolemia and in longstanding



FIG 3 19 Eruptive xanthomatosis in a man age 40 with untreated diabetes mellitus and secondary elevated cholesterol and neutral fat content of blood Eruption was generalized but most pronounced in area shown Lesions gradually disappeared within 6 months following initiation of therapy (Courtesy Dr Maurice B Struss, Chief, Medical Service, Boston Veterans Administration Hospital)

biliary or cholangiolitic cirrhosis of the liver In these disorders one is also likely to find plaques elsewhere on the skin especially the face and creases of the palms (*xanthoma planum*), elevated nodules in the skin particularly over the buttocks and extensor surfaces of the elbows (*xanthoma tuberosum*) and in the tendon sheaths especially of the Achilles tendons and the extensor tendons of the hands (*xanthoma tendonosum*)

Primary hyperlipemia often presents lesions resembling xanthoma tuberosum Depending on the level of fats in the blood, these tend to appear, enlarge, regress and even disappear in cycles (*eruptive xanthoma*)

In Hand-Schuller-Christian syndrome xanthomatous plaques may be seen on the corneas and mucous membrane of mouth or larynx Skin lesions are rare; when present they are limited to the flexor surfaces of the extremities

Diabetes and, rarely, nephrosis and myxedema may develop eruptive xanthomatosis due to secondary hypercholesterolemia.

Hemorrhage. Intracutaneous or subcutaneous hemorrhages occurring spontaneously or after negligible trauma are often important manifestations of constitutional disease Lesions less than 2 mm in diameter are referred to as



Fig 3 20 Purpuric spots and ecchymoses in symptomatic purpura (Courtesy Children's Hospital, Boston)

petechiae, from 2-5 mm. as *purpuric spots*, larger ones as *ecchymoses*. If there is sufficient extravasation of blood to produce pronounced elevation of the skin or a palpable mass the lesion is called a *hematoma*. Hemorrhagic areas are red at first, later shade into brown, then blue, then yellow and finally fade out entirely. The smaller ones do not elevate the skin or disappear on pressure as do the erythematous lesions of many exanthematous diseases and toxic disturbances Small petechiae appearing in the skin are often an important manifestation of subacute bacterial endocarditis. Purpuric spots or ecchymoses occur in many different maladies· the various types of purpura, the leukemias, lymphoblastoma, aplastic anemias, vitamin C deficiency, smallpox, typhus fever, streptococcus, meningococcus, or other types of blood stream infection, severe liver disease, and others. Ecchymoses occur after very slight trauma in some healthy women.

Edema. Edema is a collection of fluid in the subcutaneous tissues The skin is usually tense, shiny, and pits on pressure, that is, the impression made on the skin by pressure with the fingers does not immediately disappear after the fingers have been withdrawn Local areas of edema are usually the result of regional infection or trauma Edema of an extremity may be part of a generalized edema or result from local interference with the flow of venous blood or lymph Generalized edema is discussed later in this chapter

Subcutaneous Emphysema. The presence of air or gas in the subcutaneous tissues gives the region an edematous appearance On palpation one obtains a characteristic crackling sensation (*crepitation*) beneath the skin In an extremity subcutaneous emphysema is most likely due to wound infection by a gas-producing organism, in the neck to puncture wound of the trachea or spontaneous mediastinal emphysema, in the trunk to puncture wound of the lung or to infection of a surgical or traumatic abdominal wound by a gas-producing organism

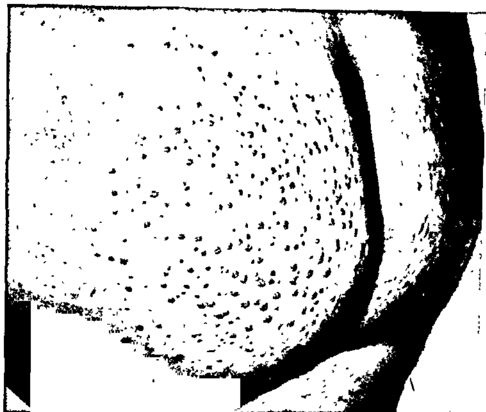


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Hemorrhage. Intracutaneous or subcutaneous hemorrhages occurring spontaneously or after negligible trauma are often important manifestations of constitutional disease. Lesions less than 2 mm. in diameter are referred to as

trunk or extremities; on the extremities most disorders favor either the flexor or extensor surfaces Distribution may be symmetrical or irregular. Acne is rarely seen except on the face, neck, back and upper chest Scabies most commonly involves the webs of the fingers, the palms, the flexor surfaces of the wrists, the axillae, the regions about the nipple, umbilicus and male genitalia, and sometimes the gluteal region, it is never seen on the face except in infants. On the extremities, psoriasis chooses the extensor surfaces; lichen planus, the flexor surfaces

Before diagnosis can be made it is often essential to know if there has been exposure to any specific infectious disease, exposure to or ingestion or inhalation of any known toxic agent, if the patient has allergic tendencies, and if there is any systemic disease or a disturbance in some other part of the body.

Eruptions of Systemic Disease

Infectious diseases rarely encountered in this country are for the most part not included here The eruptions described are important for diagnosis but alone are rarely sufficient to establish it.

Measles (Rubeola). The eruption appears first as dark red macules which rapidly increase in number and coalesce to give a blotchy appearance They vary greatly in size Later they are maculo-papular and in severe cases may become hemorrhagic. The eruption begins 1–5 days after the onset of the catarrhal symptoms and fever, gradually fading out within a week or two It



Fig 3 21 Measles, second day of eruption Macules of varying size and shape with coalescence in some areas (Courtesy Dr Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

Primary Lesions. In any dermatologic disturbance the initial localized change is known as the *primary lesion.* The forms into which it is modified by natural evolution or extraneous factors such as trauma or infection are called secondary lesions. The descriptions listed below are modeled after Swartz.[1]

MACULE. A circumscribed discoloration without elevation, such as a freckle or the earliest spots of measles

PAPULE. A solid, elevated, pathologic formation not over, roughly, 4 mm. in diameter, such as the ordinary red pimple in acne.

VESICLE. An elevated pathologic formation not larger than a papule and containing serous fluid, such as a small traumatic blister or the early lesions of chicken pox.

PUSTULE An elevated lesion the size of a vesicle but containing purulent rather than serous fluid

BULLA An elevation containing serous fluid but larger than a vesicle, such as the large blister so often seen in a burn.

NODULE A circumscribed solid mass which projects upward, downward into the deeper cutaneous layers, or both, and usually not over 1–2 cm in diameter

TUMOR A solid pathologic formation larger than a nodule

WHEAL A circumscribed elevation produced by edema in the corium and varying in size from 1 millimeter to several centimeters It is likely to be transitory.

Secondary Lesions.

SCALE An exfoliation of epidermis (peeling) Scales may be fine or coarse, brawny, silvery, dirty or white, and firmly or loosely attached

CRUST An accumulation of drying serous, purulent or sanguineous material and epithelial debris

EXCORIATION. A superficial abrasion such as a scratch mark or scrape

FISSURE. A crack in the skin usually extending through the epidermis into the corium

ULCER A circumscribed loss of substance extending through the epidermis into the corium

SCAR A connective tissue formation replacing tissue lost by a destructive process, and involving the corium or deeper layers.

PIGMENTATION. This is discussed earlier in the chapter.

The identification of a skin disorder by no means depends on the character of the lesions alone Of equal importance are the manner of onset, distribution, duration, and character and rapidity of evolutionary changes Some disturbances have an acute course, others are chronic. In some, the lesions appear simultaneously all over the body, in others, they begin in one region and spread progressively to other parts, in still others, they appear in crops, so that lesions of different ages may be present in a particular region at the same time.

Depending on the ailment, the lesions may show predilection for the face,

[1] SWARTZ, J H *Dermatology in General Practice* Baltimore, The Williams & Wilkins Company, 1953



FIG 3 23 Scarlet fever, second day of eruption. Punctate rash on trunk and extremities (Courtesy Dr Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

which appears within 12–24 hours, is a fine, punctiform eruption of small, vividly red macules which disappear on pressure The skin between them has a bright, erythematous blush. The macule may be slightly papular, giving a goose-flesh appearance, and in severe cases some of these minute papules may become vesicular. In extreme cases some are hemorrhagic. Appearing first on the neck, the eruption spreads downward to involve the arms, trunk, and legs. The flexor aspects of the extremities show more change than the extensor. The face, except for the flush, is not usually involved. If an affected area of the skin is lightly brushed with the tip of the finger, a line of blanching will appear and last 2–3 minutes (*tache cérébrale*) Pressure from clothing may cause a similar picture If somewhat firmer pressure is used the blanching will last only a half minute and be replaced by a line of increased redness This vasomotor reaction is usually seen in scarlet fever but, contrary to general opinion, is not a diagnostic sign since it occurs in similar rashes brought about by other causes In the mild cases the eruption may last only a few hours, but on the average it remains for a week or 10 days and disappears gradually. Desquamation is common It begins about a week after disappearance of the eruption, may last from a few days to several weeks, and in general is proportionate to



FIG 3 22 German measles, first day of eruption Typical macular rash on face, arms and trunk (Courtesy Dr Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

usually starts on the forehead or behind the ears along the hairline, soon involving the face and neck and spreading to the rest of the body. The face is often swollen and puffy On the extremities the lesions are more pronounced on the extensor aspects.

German Measles (Rubella). The rash is maculo papular. The spots are smaller and pinker than those of measles and the skin not as blotchy. Sometimes the appearance is scarlatiniform. The rash appears within 24 hours after onset of catarrhal symptoms, usually first on the face or neck It spreads rapidly downward, covering the entire body within 24–36 hours It fades within 2–3 days.

Scarlet Fever. With the onset of fever and sore throat the skin, especially of the face, becomes flushed and there is frequently circumoral pallor. The rash,



FIG 3 25 Chickenpox, fourth day of eruption View of back showing lesions in various stages of development—papules, vesicles, pustules, and crusted scabs (Umbilicated area is an old traumatic scar) (Courtesy Dr Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

times hemorrhagic. There are marked congestion and edema of the skin The eruption appears first on the forehead at the hairline, spreading progressively downward The abdomen, groin and legs are less affected than the upper parts, involvement of the palms and soles is profuse In contrast to chickenpox, where lesions are found in various stages of development at the same time and are not umbilicated, all smallpox lesions are in the same stage of development at the same time and do become umbilicated During the second or third week crusting begins. The scabs gradually fall off, leaving a pitted skin in all but the mild cases In the third or fourth week there is desquamation which is coarser than the fine, flaky scaling of scarlet fever.

Early Syphilis. The skin lesions of syphilis are classified as primary, secondary and tertiary The course of the disease is now divided into only two stages. the early which includes primary and secondary lesions and the late, which includes subsequent developments. Primary and secondary lesions contain *Treponema pallida* in abundance and are infectious, late lesions show very few organisms and are usually not infectious



FIG 3 24 Desquamation of legs and feet following scarlet fever (Courtesy Children's Hospital, Boston)

the severity of the illness Typically the desquamating material is fine and flaky; *it may be thick and scaly on the palms* and soles, sometimes actually exfoliative. In mild cases it is most evident on the palms and soles, or on the tips of the fingers or toes beneath the nail edges.

Chickenpox (Varicella). Mild fever and malaise may or may not precede the eruption by a day or so. The typical lesions begin as red maculo-papules which within a few hours become vesicles, surrounded by small red areolae Two or three days later they become pustular and after another day or so crusting occurs, with formation of dark scabs which gradually fall off. Mild scarring is an occasional sequela The eruption usually begins on the back or chest or on the forehead or face but soon appears on other parts of the body There is not the progressive spread from above downward seen in some of the other exanthemata Lesions appear irregularly in crops, and in a given region may be found in all stages of development at the same time

Smallpox (Variola). A mild scarlatiniform rash may appear during the prodromal stage, preceding the true eruption The characteristic smallpox lesions begin from 2–5 days after onset of the prodromes They start as red macules, 2–5 mm in diameter, which shortly become papular About the sixth day they evolve into umbilicated vesicles; after another day or so they become pustular. The lesions are usually discrete; in the severe cases they may be confluent, some-

or other antibiotic within a period of weeks before or after exposure or appearance of the lesion.

In the equivocal case of primary or secondary syphilis, the *Treponema pallidum* immobilization (*TPI*) test may establish the diagnosis However, it is not entirely dependable since it may remain negative for some time in the untreated patient Furthermore, if the test is negative at the start of treatment it may never change to positive, or if positive when treatment is initiated, it may rapidly revert to negative

The transition between the primary and secondary stages is not clearly defined The secondary manifestations appear 6–8 weeks after the primary lesion, sometimes earlier or later They may reappear at any time within 3–4 years (*relapses*). There are usually sore throat, feverishness, malaise, and perhaps headache and bone and joint pain

CUTANEOUS ERUPTION The secondary cutaneous manifestations are protean, simulating a variety of skin diseases They tend, however, to have certain characteristics in common the lesions usually have rounded, circinate forms with sharp edges, are polymorphic, show progressive development and symmetrical distribution, and do not ordinarily cause pain or itching Whenever lesions are grouped and appear in annular or ring-shaped pattern, syphilis must be suspected A macular or maculo-papular rash is the type most frequently encountered, less often, a pustular, tubercular or vesicular type, or a combination

The *macular* eruption is usually found on the trunk, flexor aspects of the extremities, and on the forehead but not elsewhere There may be definite red or ham-colored macules or merely a diffuse blush. This form may be difficult to distinguish from that of measles or German measles

The *maculo-papular* form shows general distribution but the face, palms and soles are favorite sites Scaling occurs in the later stages Mucous patches and condylomas are almost always found with this type.

MUCOUS PATCHES. See chapter 4.

CONDYLOMAS These flat, grayish, moist, fairly well circumscribed papules usually ½–1 cm in diameter are found on moist surfaces, especially where there is friction They most commonly occur about the anus and female genitalia, occasionally in the axillae, between fingers and toes, and beneath pendulous breasts

LOSS OF HAIR Chiefly affected are the temporal and occipital parts of the scalp, and the eyebrows The baldness is patchy, giving a moth-eaten appearance

LYMPHNODITIS Lymphnode involvement often appears before the cutaneous manifestations Palpation of the superficial nodes, such as the postauricular, cervical, epitrochlear, axillary and others may show some or all to be slightly enlarged and hard, but not tender

Secondary syphilides usually disappear leaving no visible evidence. As in the case of the primary lesion, *the diagnosis of secondary syphilis must be confirmed by laboratory tests* Dark-field examinations of material from secondary

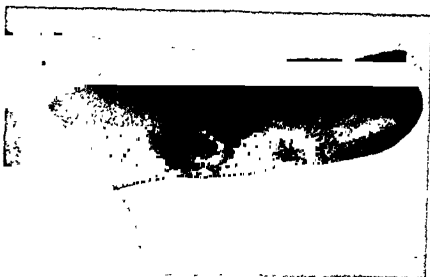


Fig 3 26 Chancre on finger

Chancre This is the primary lesion It develops at the point of inoculation It may be so inconspicuous or, especially in women, so situated as to escape notice Although chancre usually occurs on a genital organ it is extragenital in approximately 10 per cent of acquired cases, appearing most likely on *a lip, tonsil, nipple or finger* The incubation period is variable, averaging 2-3 weeks Chancre is usually single but is multiple often enough to make this unreliable as a diagnostic point It begins as a small papule which soon becomes eroded or ulcerative. It varies in diameter from 1-2 mm. to 1 cm , sometimes even larger The border is usually smooth and sharply defined, often by a hemorrhagic line, the base is smooth, indurated, and has a raw-beef color beneath the superficial crusting Characteristically, there is a yellow, serous exudate On the tongue or female genitalia, the lesion may appear only as an erosion. Chancre is usually painless, indolent, lasts from 3-6 weeks, and unless secondarily infected, heals spontaneously, leaving a superficial scar Regional lymphnodes are always enlarged and hard They remain discrete and do not suppurate unless there is secondary infection

The diagnosis of chancre cannot be made on the basis of its physical characteristics alone Confirmation by dark-field examination of exudate or by blood studies is essential. Unless the initial dark-field smear is positive, this test must be repeated at intervals until the lesion heals Examination of material from lesions in the mouth must be cautiously interpreted, only an experienced observer can differentiate *Treponema pallidum* from other spirochetes that may be present If diagnosis cannot be established by the dark field, one must rely on blood findings Negative serologic tests are not dependable in the early stages Often the response does not become positive until several weeks after the chancre has developed so that in pertinent cases tests must be repeated for *several months* This is particularly true if the patient has received penicillin

or other antibiotic within a period of weeks before or after exposure or appearance of the lesion.

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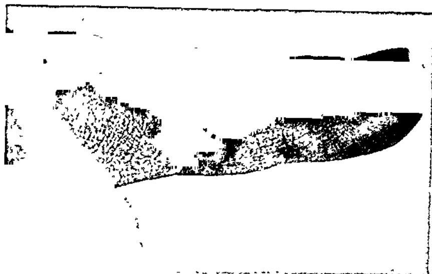


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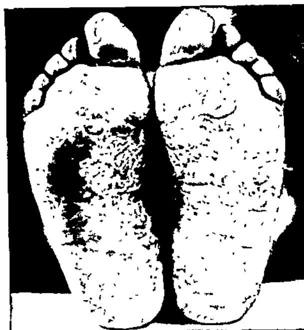


FIG 3 28 Eruption and scaling of soles in early syphilis Similar changes may be seen on hands.

lesions, preferably mucous patches or condylomas may be sufficient. If these are negative one must then depend on repeated serologic and/or TPI tests, but here too, the issue may be confused for months by recent administration of most antibiotics.

Late Syphilis. A variable interval—months or years—elapses between the time of infection and the appearance of tertiary manifestations. Late lesions are



FIG 3 29 Alopecia in early syphilis



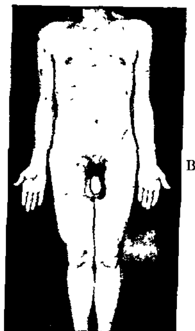


Fig 3 27 Early syphilis Generalized discrete maculo-papular eruption

A *Note especially involvement of left angle of lips and cutaneous margin of right nostril* A lesion at either of these sites, encountered in an eruption of this type, emphasizes the possibility of syphilis

B Involvement of palms and penis with this type of eruption also emphasizes the possibility of syphilis (Courtesy Dr Maurice B Strauss, Chief, Medical Service, Boston VA *Hospital*)

the TPI test will probably clarify the problem It is virtually always positive in the patient with untreated late or latent syphilis; in one who has received therapy it reverts to negative slowly.

Congenital Syphilis. The cutaneous manifestations are very similar to those of the secondary stage of acquired syphilis. They rarely appear before the third week of life The eruption is usually papular. The chin, circumoral region, palms, soles and anogenital region are the sites of predilection. Mucous patches and condylomas are usually present. Deep fissures (*rhagades*) radiating outward from the borders of the lips and corners of the mouth are often seen; when healed, they leave characteristic linear scars What was stated in the preceding paragraph regarding the TPI test in late or latent syphilis applies also to the congenital form.

Tuberculosis. Tuberculosis of the skin is a chronic disorder. It occurs in various forms. Dermatoses showing microscopically the characteristic tissue reaction of the disease are:

LUPUS VULGARIS, consisting of yellowish-brown or reddish brown nodules usually 1–5 mm. in diameter which tend to ulcerate and result in atrophy and scar formation. The disease progresses by the appearance of new lesions peripherally, but new lesions also occur in the scar tissue, a feature which distinguishes lupus from late syphilitic and other destructive processes. Lupus is most common on the face It may involve the mucous membranes

SCROFULODERMA, an area of indolent and sluggish ulceration of the skin overlying a tuberculous lesion beneath and connected with it by a discharging sinus. Scrofuloderma is most common in children, and most often seen on the neck as a result of tuberculous lymphnoditis or an extremity as a result of a tuberculous joint.



FIG 3 31. Lupus vulgaris



Fig 3 30 Ulcero nodular lesions of late syphilis Areas of atrophy and pigmentation due to healing interspersed between active lesions (Courtesy Dr Earl A Glicklich)

almost always painless They are few in number, all of the same general character, and not symmetrically distributed For the most part the lesions have a definite circinate configuration, being arranged as segments of circles but never forming complete circles They are usually nodular, indurated and indolent, with a tendency to extend peripherally while healing centrally and to show increased pigmentation in the healing areas In contrast to granulomas due to other causes, activity never recurs in the scar tissue They may involute, leaving atrophy and pigmentation but are more apt to ulcerate. The ulcers characteristically have a sharply defined border, giving them a punched-out appearance. Ulcerating lesions also show a tendency to heal in one part while extending elsewhere.

Single gumma is a form of late syphilis seen occasionally It starts as a nodule, sometimes growing to tumor size. It is especially apt to occur at the site of trauma It usually breaks down, leaving a sluggish area of ulceration

Late syphilides must be distinguished from the lesions of tuberculosis, leprosy, blastomycosis, sporotrichosis, coccidioidomycosis, other granulomatous disease and malignant disease. The serologic test is almost always diagnostic but in late cases a negative test does not absolutely exclude syphilis. If the clinical picture is not clear, or positive serologic tests are regarded as possibly biologically false,

more than a dozen being present at one time. They are found chiefly on the abdomen, the lower part of the chest and on the lower part of the sides, but when exceptionally profuse may also be found on the back and thighs. They disappear on light pressure but immediately reappear. They darken with age and fade out within 1–5 days. An important diagnostic feature is the alternation of successive outcroppings with intervals during which the skin is clear.

Typhus Fever. The eruption usually appears around the fifth day. The typical lesions begin as round or oval, reddish or pink macules, 2–5 mm. in diameter. The rash begins on the chest, abdomen and back, and soon spreads to the extremities and sides of the neck. The face, palms and soles are usually spared, in contrast to Rocky Mountain spotted fever, in which these parts are almost invariably involved. In mild cases, the eruption is scanty. The typical lesions are not well-circumscribed but do not, as a rule, coalesce. On pressure they fade but do not disappear, about the ninth day or earlier, they darken and show no fading on pressure. There is no tendency for them to appear in crops, no new ones developing after the second or third day of the eruption. After recovery, the spots, which have darkened to brown, may persist for weeks. In severe cases the eruption is extensive and often hemorrhagic; gangrene of the tip of a digit, an earlobe, the nose or the scrotum or penis may occur. The Weil-Felix agglutination reaction is usually but not necessarily positive with the OX-19 strain of proteus bacillus after 10–12 days.

Tsutsugamushi disease (scrub typhus), prevalent in eastern and southern Asia, parts of Australia and many of the Pacific islands, shows an eruption similar to that seen in epidemic and murine typhus but likely to be evanescent. Often seen at the point where the infecting mite was attached is an eschar—a raised, round or oval lesion 1 mm–1 cm. in diameter. It is red except for a black, necrotic center which may slough off, leaving a small ulcer. By the end of the second week, the Weil-Felix agglutination reaction may become positive with the OX-K strain of proteus bacillus but it is negative with OX-19 and OX-2.

Rocky Mountain Spotted Fever. The rash usually appears on the third to sixth day. At onset the lesions are rose-colored macules 1–5 mm. in diameter, which disappear on pressure. Later they darken to a deep red or purplish hue and no longer disappear when pressed. In contrast to typhus fever, the wrists and ankles are involved first but within 24–36 hours the eruption will also appear on the rest of the extremities, the forehead and trunk. In severe cases, the spots may be petechial or hemorrhagic. Fading usually begins about the end of the second week, although traces remain for some time. After the second week the Weil-Felix agglutination reaction may become positive with the OX-19 or OX-2 strain of proteus bacillus or both. Both occasionally remain negative.

Acute Meingococcus Infection. The predominant focal manifestations are meningitis, an eruption and inflammation of one or more joints. These appear in various combinations. When meningitis does not develop, as often happens, particularly in an epidemic, the eruption may be the most important early clue to diagnosis. In general, the severity of the rash is proportionate to the severity of the infection yet the meningitic form may occur without a striking

Dermatoses failing to show the characteristic tissue reactions of tuberculosis but thought to be caused either by tubercle bacilli of attenuated virulence or by an allergic response to a tuberculous focus elsewhere are:

PAPULONECROTIC TUBERCULIDS, firm, isolated papules, 2–5 mm in size, which appear in crops, develop necrotic centers and heal spontaneously with pitted scarring. The sites of predilection are the face and upper extremities, sometimes the buttocks. The extensor surfaces are favored They are most likely to occur in children and young adults

ERYTHEMA INDURATUM, characterized by violaceous nodules, 5–15 mm in diameter or even larger, which ulcerate, leaving depressed scars. Seen most frequently in women, the lesions appear characteristically on the legs, chiefly the calves, and are worse in winter.

LICHEN SCROFULOSORUM, consisting of grouped, slightly red follicular papules which usually appear on the lower two-thirds of the back It almost always occurs in children

Some observers also place other lesions such as those of sarcoidosis, lichen nitidus and granuloma annulare in this group but such a classification is open to question.

Acute Rheumatic Infection. A characteristic skin eruption (*erythema marginatum*) is not infrequently seen during an attack of active rheumatic infection It is a form of erythema multiforme The lesions are pale pink, circinate macules which spread peripherally They vary in size from 2 cm to conglomerate areas 10–20 cm in diameter or even larger The trunk and flexor surfaces of the forearms and thighs are the sites of predilection The eruption is transient, it may last from a few minutes to one or two days *It tends to recur especially* after a bath or exposure to the sun

Typhoid Fever. Discrete rose-red, oval or rounded macules or maculo-papules, averaging 2–4 mm in diameter, (*rose spots*) *usually appear toward the end* of the first or second week of the disease Rose spots are rarely numerous, not



FIG. 3 32. Erythema marginatum of forearm in acute rheumatic infection

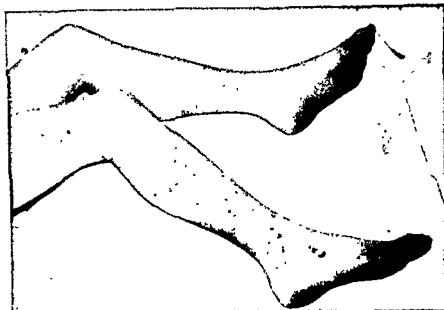


FIG 3.34 Eruption on legs in a soldier age 19 with acute meningococcus infection Patient had fever and joint pains but no indications of meningitis

episodes, the patient may be symptom free or show evidence of smoldering bacteremia An attack of acute meningitis may develop at any time.

Epidemic Hemorrhagic Fever. Unknown in this country but prevalent in Korea where it has been frequently encountered in United Nations military personnel, this is a serious acute infectious disease, the cause of which has not been determined Onset is usually abrupt with rapid development of chills, high fever, headache, restlessness, insomnia, coryza, cough, anorexia, nausea and vomiting, and joint and muscle pains. The acute phase is usually over within a week or 10 days The hemorrhagic manifestations, starting about the third day, can involve the skin, conjunctivas, mucous membranes or any of the internal organs They range from petechiae to large areas of ecchymosis or gross hemorrhage. Bleeding from gums, nose, lungs, kidneys or gastrointestinal tract is a frequent and serious complication Renal impairment simulating lower nephron nephrosis is common In the serious cases, early death is likely from hemorrhage or shock

Erysipelas. Erysipelas is an acute specific infection of the skin and subcutaneous tissue, probably caused by *Streptococcus hemolyticus* It is usually accompanied by marked systemic disturbance. The most common location is the face but other parts may be involved, particularly the zone surrounding an ulcer or the site of recent injury or operation. The lesion begins as a small area of swelling, redness and tenderness that spreads rapidly in all directions The edges of the infected zone are raised and sharply demarcated from the surrounding normal skin; centrally vesicles or small blebs often appear. The



FIG. 3 33 Eruption on arms and trunk in Rocky Mountain spotted fever (Courtesy Dr Robert N Ganz)

rash In the typical case of meningococcic infection with meningitis, the eruption and the cerebrospinal manifestations appear at about the same time—within 48 hours following onset of systemic symptoms The rash may be general or largely confined to the extremities The spots are dusky-red, usually petechial but sometimes maculo-papular, have a diameter of 1–5 mm and do not disappear on pressure; after 3–4 days they become brownish and begin to fade.

Mild or moderate acute meningococcemia without meningitis shows a scanty eruption, macular or maculo-papular, and confined to the distal parts of the extremities, especially the wrists, ankles, palms and soles

In the severe, fulminating cases with or without meningitis, the rash is profuse, general and usually petechial or purpuric It develops with amazing rapidity, often during a few minutes' observation new lesions can be seen to appear. The spots sometimes merge, giving large areas of ecchymosis which can become gangrenous Meningococci may be demonstratable in a stained smear of tissue fluid obtained from a lesion Meningitis or arthritis may appear but often the patient dies of overwhelming infection before such focalization occurs.

Chronic Meningococcus Infection. In this less malignant form of meningococcic infection, there are intermittent episodes of fever, rash, and usually mild or moderate arthritis. The eruption is rarely intense, likely to be maculo-papular, and limited to the distal parts of the extremities Occasionally, lesions resembling those of erythema nodosum appear on the extremities Between

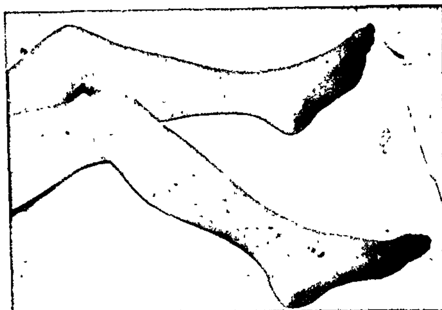


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rash In the typical case of meningococcic infection with meningitis, the eruption and the cerebrospinal manifestations appear at about the same time—within 48 hours following onset of systemic symptoms The rash may be general or largely confined to the extremities The spots are dusky-red, usually petechial but sometimes maculo-papular, have a diameter of 1–5 mm and do not disappear on pressure; after 3–4 days they become brownish and begin to fade

Mild or moderate acute meningococcemia without meningitis shows a scanty *eruption, macular or maculo-papular, and confined to the distal parts of the extremities*, especially the wrists, ankles, palms and soles

In the severe, fulminating cases with or without meningitis, the rash is profuse, general and usually petechial or purpuric It develops with amazing rapidity; often during a few minutes' observation new lesions can be seen to appear. The spots sometimes merge, giving large areas of ecchymosis which can become gangrenous. Meningococci may be demonstratable in a stained smear of tissue fluid obtained from a lesion Meningitis or arthritis may appear but often the patient dies of overwhelming infection before such focalization occurs.

Chronic Meningococus Infection. In this less malignant form of meningococcic infection, there are intermittent episodes of fever, rash, and usually mild or moderate arthritis. The eruption is rarely intense, likely to be maculopapular, and limited to the distal parts of the extremities. Occasionally, lesions resembling those of erythema nodosum appear on the extremities. Between

ation which fails to respond to the usual treatment. This complication was common in troops in the tropics Infection occurs not only in a deep wound but is frequent in minor lesions such as insect bites or superficial abrasions It may also be superimposed on some other skin disorder such as dermatophytosis or impetiginous dermatitis. The typical lesion, occurring singly or multiply, is a rounded punched-out ulcer varying in diameter from a few millimeters to several centimeters, with a rolled, slightly raised, sometimes undermined edge. In the early stages, the base is covered by a yellowish or brownish-gray membrane which, if peeled off, reveals a clean hemorrhagic base. Within a few days a black or brownish adherent eschar usually forms and may remain for several weeks; if present, this is an important diagnostic clue. In later lesions, the base is usually covered with a grayish-brown, moist exudate. Another characteristic is that, although the early lesion is painful and tender, the base of the late lesion is likely to be anesthetic and painless. Healing begins peripherally, progressing evenly toward the center. The scar of the healed lesion is clean and atrophic, likely to be depressed and sharply demarcated from normal skin. Vesicular and bullous eruptions resembling pemphigus or dermatitis herpetiformis have been reported. The diagnosis should be confirmed by cultures; recovery of the organism, especially from an older lesion, is often difficult, requiring repeated, thorough bacteriologic studies. If the disease is unrecognized, the patient may develop faucial diphtheria, diphtheritic neuritis or myocarditis. Spread of infection to contacts is an added hazard. The diagnosis is too often overlooked until some such development suggests it.

Tularemia. The primary lesion occurs at the site of inoculation. Because the disease is so often acquired from handling an infected rabbit or other animal, it is frequently found on a hand, finger or forearm In tick-borne infection, which occurs more frequently than is generally thought, the primary lesion may appear anywhere on the body, most commonly a lower extremity, buttock or the lower abdomen It appears as an indolent ulcer from 0 5–3 cm or more in diameter with a punched-out center, raised, irregular edges, and a surrounding zone of erythema and induration Regional lymphnodes become swollen, tender and usually suppurate. A generalized eruption is not uncommon, appearing from 5 days to 2 weeks or more following the primary lesion The rash, red and usually maculo-papular, but sometimes vesicular or pustular, is diffuse but likely to be more pronounced on the extremities and upper part of the trunk.

Glanders. A generalized infectious disease found chiefly in persons working with horses, glanders is not common in this country. The site of inoculation may be a mucous membrane or a break in the skin The cutaneous lesions appear at the site of inoculation, elsewhere on the body surface and on the mucous membranes Beginning as small, subcutaneous nodules (*farcy buds*), they rapidly break down and give rise to foul ulcers. These tend to spread, involving extensive areas and, on the mucous membrane, give rise to mucoid

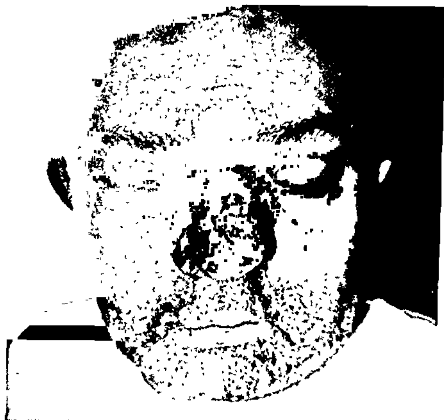


FIG 3 35 Erysipelas, severe, with ulcero gangrenous lesions on nose due to secondary staphylococcus infection (Courtesy Dr Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

skin is tense and shiny If the disease involves the orbital region or other areas where subcutaneous tissue is loose, edema is pronounced The process is usually confined to one region, rarely extending to other parts. With improvement, *the inflammation disappears and the skin resumes its normal appearance*

ERYSIPELOID. A cellulitis-like inflammation caused by the erysioplothrix of swine erysipelas, this presents a picture resembling erysipelas In contrast to the latter, it is typically seen on a hand, resulting from a scratch while handling *infected fish, crab, chicken or, less commonly, some other infected animal* The systemic reaction is not severe

Diphtheria. Secondary infection of an existing skin lesion by *Corynebacterium diphtheriae* is a serious possibility that must be excluded whenever diphtheria *is known to be present in the community and in any case of indolent ulcer-*

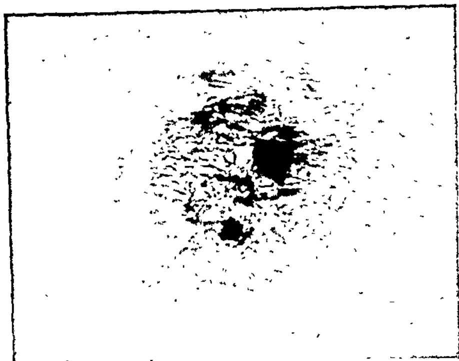


FIG 3 37 Anthrax Primary lesion (malignant pustule) on upper abdominal wall Note ring of edema surrounding black eschars Patient was a wool sorter who worked stripped to the waist (Courtesy Dr Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

or mucopurulent viscid discharge In the acute cases, the systemic reaction is severe and the lesions numerous Diagnosis is confirmed by microscopic examination of material from a lesion.

Anthrax. This is an acute, infectious disease of animals sometimes communicated to human beings, especially those who work with farm animals, wool, hides, hair or bristles An improperly sterilized shaving brush is an occasional source of infection. The cutaneous lesion appears at the site of inoculation, usually an exposed surface such as the neck, face or hand Slight burning or itching of the area precedes the appearance of a reddish papule which rapidly becomes vesicular and spreads to form a dark brown or black eschar, 0 5–2 cm in diameter, covered and surrounded by small groups of vesicles Edema of the surrounding tissues is marked and spreads to include a considerable area, this extensive infiltration is a characteristic feature. If recovery occurs, the central gangrenous area will slough and leave a deep scar.

Plague In plague a petechial or hemmorhagic, less often pustular, eruption may appear

Diabetes. Single or multiple non-symmetrical plaques averaging 2 cm. in diameter but often smaller or much larger may occur in diabetes (*necrobiosis*



A



B

Fig 3 36 Tularemia, primary lesion

A On abdominal wall following tick bite (Courtesy Dr Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

B On finger following handling of infected animal (Courtesy U. S Public Health Service)

haps atrophy and pigmentation Involvement is variable. The changes are most likely to appear on the extremities and face but may involve the neck, chest, shoulders and sometimes most of the body (see Chap 35).

Scleroderma. The skin and subcutaneous tissues are thickened, brawny and dry, especially on the hands and fingers, toes, forehead, neck, face, and chest Raynaud's phenomena of the extremities are common and may end, especially on the tips of the fingers, in necrosis and ulceration (see Chap 35).

Cutaneous eruptions, most commonly of the maculo-papular variety, but often taking some other form, are also encountered in other infectious diseases such as brucellosis, mononucleosis, rat-bite fever, Haverhill fever and relapsing fever, in certain blood dyscrasias and many toxic and allergic states

Diseases of Fungus Origin

Actinomycosis, sporotrichosis, blastomycosis, coccidioidomycosis, moniliasis and other fungus diseases show cutaneous lesions. These are for the most part granulomatous and slowly progressive. They are characterized first by formation of nodules in the skin which may develop into cold abscesses and later break down, forming areas of chronic ulceration, sinuses and scars. An allergic skin eruption of one type or another may also appear.

Tumors of Skin and Subcutaneous Tissue

Fibroma. A benign connective tissue tumor occurring singly or multiply in the subcutaneous tissue, fibroma may be flat, sessile, pedunculated, white or pinkish in color and either soft or hard.

Multiple Neurofibromatosis (Von Recklinghausen's Disease). Tumors originating in the nerve sheaths arise deeply or superficially and usually appear in large numbers They have the same physical characteristics as fibromas Sometimes a tumor developing in the sheath of a periosteal nerve will invade underlying bone and simulate a true bone tumor.

Lipoma. Here one finds a soft, rounded, subcutaneous tumor varying in diameter from 0 5–8 cm. or more, and somewhat movable It is fairly well circumscribed yet tends to merge into the surrounding normal tissue. It is often multiple.

Verruca (Wart). This is a small circumscribed epidermal growth of varying size which occurs singly or multiply It is at first usually smooth, later roughened and papillated It may be flat or domed, filiform or acuminated. *Verruca vulgaris* is the common wart; it is most often found on a finger or hand and is likely to be multiple. *Verruca plantaris*, occurring on the sole of the foot, is hard, tender and painful on walking *Verruca acuminata (venereal wart)* is described in chapter 33 Warts are now generally considered due to local infection, probably virus

Nevus (Mole). Hypertrophy of one or more of the cutaneous structures results in a circumscribed area of new growth usually 1–3 mm but sometimes as much as several centimeters in diameter and occasionally even larger.

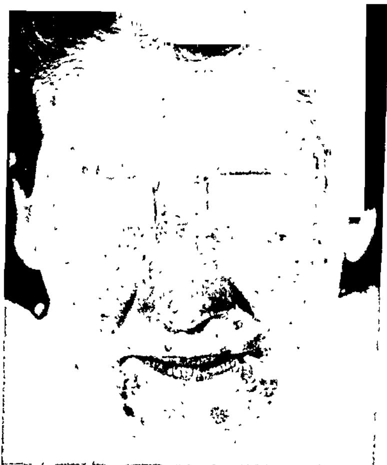


Fig 3 38 Eruption on nose and other parts of face in acute disseminated lupus erythematosus (Courtesy Dr Earl A Glicklich)

lipoidica diabeticorum) These lesions are most frequent over the legs, especially the shins The affected areas are yellowish-pink and threaded with minute blood vessels The skin is atrophied and depressed *Ulceration is rare*

Disseminated Lupus Erythematosus. The initial skin lesion usually appears on the malar eminences or nose as an erythematous patch which spreads fairly rapidly, eventually involving the two cheeks and the bridge of the nose in such fashion as to give the so-called butterfly appearance. The face, ears, neck and upper extremities may become involved There appears to be a predilection for exposed surfaces but other parts are also vulnerable. Although the erythematous patch is most characteristic, a wide variety of other skin lesions, most likely those seen in urticaria, erythema multiforme and erythema nodosum, *may occur simultaneously or in succession Petechiae with or without larger* hemorrhagic lesions are sometimes seen The mucous membranes may show sharply defined patches or erythema often followed by ulcerations (see Chap 35)

Dermatomyositis. The skin shows redness, scaling, thickening and later per-

tumor of skin cancer and potentially dangerous, especially if subjected to irritation, or when the lesion is near an eye or on a lip or at a muco-cutaneous junction Disc-like thickening, induration and rapid growth are danger signals.

Epidermoid Carcinoma. This is the most common malignant tumor of the skin. Likely sites are the dorsum of the hand, the nasolabial fold, lip, helix of the ear, and scalp It begins as a small red or pearly-white, hard, scaly papule or as a subcutaneous nodule. It is particularly apt to occur in an area of keratosis or a scar from a burn or some chronic skin disease such as lupus vulgaris or lupus erythematosis It soon develops into an ulcerating lesion with an adherent crust. If the latter is removed, one observes an indurated, usually papillary, base with a serous or serosanguineous discharge and a fairly well-circumscribed, often rolled border, which is pearly-white and also indurated The lesion grows quite rapidly, invading surrounding tissues. Occasionally it remains superficial or becomes a raised tumor As a late development, it metastasizes to the regional lymphnodes

Basal Cell Carcinoma. Starting as a small, glistening pearly nodule 1–2 mm. in diameter, basal cell carcinoma grows very slowly over a period of years and eventually ulcerates, showing an encrusted center and a pearly, elevated, not *markedly indurated border (rodent ulcer) Usually the lesion measures* 0.5–2 cm but may be larger The face is its most common site but it may develop anywhere on the body surface Basal cell carcinoma cannot be positively distinguished from epidermoid carcinoma except by histologic examination. It is not ordinarily malignant but transition to epidermoid carcinoma may occur at any time.

Melanoma. This tumor usually arises from a pigmented or non-pigmented mole and is as likely to occur in a small as in a large one. It may also develop in apparently normal skin, in the retina or iris It is also known as *nevocarcinoma, melanocarcinoma,* or *melanosarcoma,* the different names being due to confusion regarding the nature of the cellular constituents In its earliest stage it cannot be differentiated from a non malignant mole except by histologic examination. Transition from benign to malignant status is indicated by

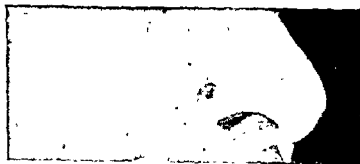


Fig 3 40 Basal cell carcinoma on nose (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)

There are several types, the most important of which are the pigmented the vascular. *Pigmented* moles are localized areas of increased pigmenta brown or bluish- or greenish-black, flat or raised, smooth or warty. Some s abnormal growth of hair. *Vascular* moles (*hemangiomas*) may be red, bl or purplish-red, smooth and level with the skin, or raised and lobular. If pressed, they show a tendency to blanch. The so-called port-wine mar birthmark is a congenital form of vascular nevus It may be small, but s times involves an extensive area of skin and subcutaneous tissue Its common site is the face.

Any mole which has been subjected to irritation or which shows increas size, change of color, crusting, bleeding, ulceration or development of nodules within the lesion or in nearby skin is likely to be malignant is particularly true of a pigmented mole, especially one which is bluish greenish-black but absence of pigmentation does not preclude the possib of malignant change if the lesion is irritated.

Keratosis. Frequent in older age groups, keratosis is a local area of hy trophy occurring singly or multiply and most commonly on exposed surf especially the face, ear, scalp and dorsum of the hands It is sharply circ scribed, dry, flat or slightly elevated and may be gray, brown, yellowis brownish-black Scaling is common, when, as often happens, a scale separ a reddish, slightly roughened area of skin is exposed It is a frequent f



Fig. 3.39 Epidermoid carcinoma, inner canthus. (Courtesy Pondville Hospital, Massachuse

runner of skin cancer and potentially dangerous, especially if subjected to irritation, or when the lesion is near an eye or on a lip or at a muco-cutaneous junction. Disc-like thickening, induration and rapid growth are danger signals.

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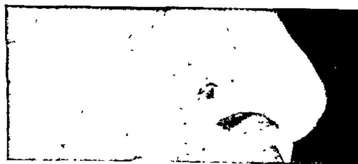


Fig 3 40 Basal cell carcinoma on nose (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)

increase in size, alteration in color or any of the other changes described above (see Nevus) As it grows, the tumor may become papillary, fungoid or extensively ulcerated Widespread metastases occur early and grow rapidly, involving almost every part of the body especially the skin near the primary lesion and the liver, brain, and lungs An occasional feature of the late stage when metastases are widespread is diffuse generalized pigmentation of the skin

A non-pigmented type of malignant sarcoma occasionally occurs as a pea-sized or somewhat larger cutaneous nodule

Sarcoma. Primary malignant tumors of mesodermal origin are encountered in the skin but are rare. As a rule they are slowly growing and locally invasive, non-ulcerative and show less tendency to generalized metastasis than other forms of skin tumor.

Metastatic Malignant Disease. Metastatic lesions of carcinoma, lymphoma and leukemia often appear in the skin, taking the form of nodules of varying size and consistency Ulceration is not uncommon Histologic examination may be necessary for diagnosis but the nature of the nodules can be inferred if diagnosis of the primary lesion has already been made Lymphoma and leukemia may also give rise to a variety of non-specific lesions of the skin which on examination show none of the characteristics of the specific secondary lesions of these diseases They may resemble psoriasis, eczema, or almost any form of dermatosis.

MYCOSIS FUNGOIDES, thought to be lymphoma of the skin, almost always—before the stage of tumor formation—passes through a so-called premycotic stage resembling one or more of the dermatoses and characterized by persistent, intractable itching The association of such skin lesions or unexplained itching with lymphnodopathy should always suggest the possibility of lymphoma

DISTRIBUTION OF HAIR

Abnormal growth or distribution of hair is in many instances probably of endocrine origin although the basic disturbance is not definable. In women adrenogenital syndrome or arrhenoblastoma of the ovary is suggested by excess hair on the face, trunk or extremities, especially when it is associated with other indications of glandular dysfunction such as recession of the temporal hair line, enlargement of thyroid cartilage with resultant deepening of the voice, enlarged clitoris and, if the disorder begins before puberty, failure of the breasts to develop normally

In males, lack of normal recession of the temporal hair line suggests primary or secondary hypogonadism Scantiness or slow growth of axillary hair, indicating low ketosteroid production, is seen in myxedema, Addison's disease and panhypopituitarism. In myxedema one also finds thinning of the eyebrows, especially their outer thirds, and thinning with increased dryness of the hair on the head In males with cirrhosis of the liver, loss of hair on the chest, and to some extent on the abdomen, is often observed

Normal adolescent males may show female distribution of pubic hair for several years after puberty but develop the typical masculine pattern later.

Persistent female distribution in males or male distribution in females, although often normal, may call for endocrine study, especially if any other features of glandular dysfunction are apparent.

WEIGHT

Average Weight

Charts indicating the normal weight for persons of a given height, age, sex and race are useful merely as guides. A weight 10–15 per cent or more above or below the given standard is not necessarily abnormal. Differences in weight between persons of the same age, sex, height, and race are controlled by the relationship between intake of food to output of energy, the size of the bony framework, heredity, temperament, and, in a small percentage of cases, differences in the activity of certain endocrine glands.

Broadly speaking, a male 5 feet in height should weigh approximately 110 pounds For each added inch there should be an additional 5 pounds, so that a man 5 feet 6 inches tall, for example, should weigh in the neighborhood of 140 pounds In females the figures are slightly lower.

The normal weight of children is as follows:

Height	*Weight*
20 inches	7 pounds
25 inches	14 pounds
30 inches	21 pounds

After the first year, the height should increase 2½ inches yearly up to the age of 11, a 12-year-old boy should measure approximately 5 feet. A 2-year-old boy should weigh 28 pounds and then gain 4½ pounds yearly, to puberty. As in adults, the figures for female children are slightly lower.

Recent Loss or Gain. Weight change is of more significance than overweight or underweight of long standing and except when relatively slight calls for careful investigation In the treatment of some disturbances, accurate day-to-day weight determinations are important; in this event, the weight should be taken before breakfast and following micturition and bowel movement in order to exclude daily variations related to the amount of fluid and solid matter in the stomach, bladder and rectum Physiologic retention of body fluid in most normal women causes gradual gain in weight averaging 1–3 pounds during the week prior to each menstrual period; there is a drop to the normal level within a day or so after its onset.

Unless one bears in mind that change in weight may be due to increase or decrease, not of flesh but of body fluid, he may in certain disorders such as congestive failure or malnutrition overlook loss of flesh because the weight remains stationary or increases as a result of abnormal fluid retention.

Gain of Weight

Obesity. When not due to normal growth or fluid retention, weight gain is almost always the result of habitual ingestion of more calories than are necessary for daily energy requirements There is marked variation in the

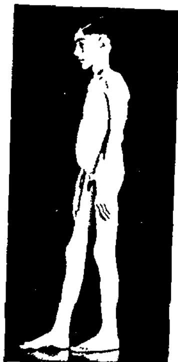


FIG 3 41 Emaciation, ascites, and dependent edema of legs due to nutritional deficiency.

ease and rapidity with which different persons, all apparently healthy, can gain weight This variation is governed by such factors as differences in expenditure of nervous and physical energy, emotional status, occupation, economic status, smoking habits and family eating habits Persistent over-eating is often a response to some emotional conflict

Edema. In many cases of cardiac, hepatic or renal insufficiency, in starvation, deficiency states, anemia and certain other disorders, serous fluid accumulates in the tissue spaces, serous cavities, or both. When retention is slight, its only manifestation is gain in weight This may lead to a mistaken impression of improvement; actually it is an unfavorable sign

Pregnancy. Gain in weight is always a feature of normal pregnancy, but in the early months it may be preceded by loss of several pounds

Endocrine Disturbance. Myxedema and some forms of pituitary or, less often, other endocrine dysfunction are sometimes responsible for gain in weight Contrary to popular belief, most cases of excess weight are not due to endocrine disease

Loss of Weight

Old Age. The degenerative change associated with the aging process result in gradual or sometimes fairly rapid diminution of body weight.

Inadequate Diet. Here we include cases of insufficient caloric intake, deficiency of vitamins or other important dietary elements because of poverty, prolonged lactation or dietary fetishes.

Loss of Appetite. This is common in most diseases. It also occurs with nervousness, worry, agitated or depressed states, alcholism, drug addiction and chronic fatigue.

Inability or Unwillingness to Eat. Such disorders as an inflammatory lesion in the mouth or throat, improper dentures, impairment of the deglutition reflex, and esophageal obstruction may make it painful or impossible for the patient to eat.

Loss of Sleep. This may be an important factor in the emaciation associated with painful illnesses Although worry is a common cause of sleeplessness, it may be responsible for loss of weight even when sleep and appetite are not affected.

Disturbances of Digestion. These may be of structural origin, such as peptic ulcer, malignant disease of the intestinal tract, colitis, dysentery, acute or chronic liver disease, or of functional origin such as persistent nausea, vomiting or diarrhea associated with a disturbed emotional state.

Increased Metabolism. In febrile states, thyrotoxicosis and leukemias, loss of weight may occur even though caloric intake is increased

Endocrine Disorders. In addition to thyrotoxicosis one finds loss of weight in diabetes and Addison's disease Contrary to general opinion, panhypopituitarism ordinarily does not show appreciable weight loss; many cases in which it is thought to be due to panhypopituitarism are probably anorexia nervosa.

Malignant Disease. Malignant disease, especially when metastases are widespread, is a common cause of weight loss. The underlying mechanism is not understood; toxic absorption may be the important factor.

Chronic Cardiovascular or Cardionephritic Disease. Loss of flesh is common in the late stages but weight may remain constant or increase as a result of edema.

Chemical Poisoning. Absorption of certain industrial chemicals, such as lead, may lower the weight

Blood Disease. In leukemia the elevated metabolic rate is probably the cause In some cases of anemia poor nutrition may result in loss of weight but in others it is minimal or absent

Neurologic Disease. Loss of weight occurs in the late stages of many neurologic disorders, especially dementia paralytica, tabes dorsalis and Parkinson's disease.

Loss of Body Fluid. Common causes are lowered intake, persistent vomiting, sweating, diarrhea, diuresis and hemorrhage

TEMPERATURE

There is no absolute normal Body temperature varies a degree or more in different persons and from time to time in the same person. It is generally lower during sleep and increases by as much as a degree (F.) with activity. As a general rule, an oral temperature above 97°F. and below 99°F. is regarded as normal. In a healthy person, a reading of a few points below 97°F. on arising is of no significance During the first half of the menstrual cycle the normal

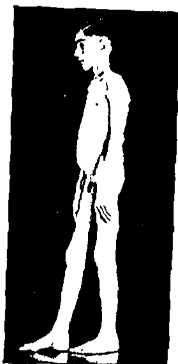


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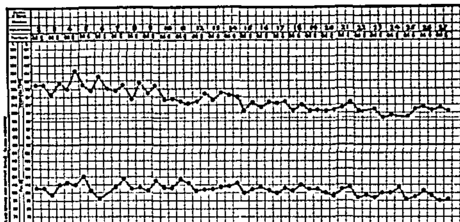


FIG 3 42 Continued fever with termination by lysis Chart of later weeks in a case of typhoid fever

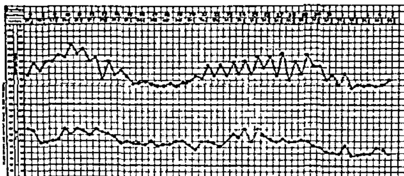


FIG 3 43 Relapsing fever in a case of brucellosis

orrhage or other intracranial insult, or coma from any cause Nervous excitement will occasionally produce a mild rise in temperature

4 Hemorrhage Following an initial drop in temperature, a moderate fever, the cause of which is not clear, is often present for a few days after a severe hemorrhage

Continued Fever. The temperature does not return to normal at any period during the 24 hours. This is seen in many cases of typhoid fever, pneumonia, tuberculosis and other infectious diseases

Intermittent, Hectic or Septic Fever. The temperature drops to a normal or subnormal level once or more in 24 hours This occurs in double tertian malaria, sepsis and sometimes in tuberculosis and late malignant disease.

Relapsing Fever. Fever is of days' or weeks' duration with alternating periods of apyrexia This type is seen characteristically in brucellosis, lymphoma and the spirochetal disease, *relapsing fever*

Barring some long infectious disease such as typhoid fever, the most likely

woman has a slightly lower body temperature than during the second half. If her temperature is accurately taken each morning throughout her cycle an abrupt rise of several tenths of a degree, sometimes preceded by a drop on the previous day, will be observed at about midcycle; it will remain at the higher level until just prior to the subsequent period when it will fall to the lower level characteristic of the first half. This midcycle rise is presumed to indicate the day of ovulation.

Oral Observation. Under ordinary circumstances, temperature is taken by mouth. The thermometer is held for at least 3 minutes under the patient's tongue; the lips must be kept closed False readings are often obtained when hot or cold substances have recently been in the mouth, when the patient has recently been breathing cold air, if he breathes through his mouth while the temperature is being taken, or if the thermometer is left in the mouth for too short a time Inexperienced persons often obtain incorrect readings by failing to shake down the mercury or by washing the thermometer in hot water before using it. When the patient is hysterical or thought to be a malingerer, he must be closely watched while his temperature is being taken, lest he shake the mercury upward, apply heat to the bulb, or otherwise attempt deception

Rectal Observation. The temperature of the rectum normally averages 1°F. higher than that of the mouth. Rectal observations are more accurate than oral and should always be taken when it is important to detect slight fever, as for example, in a patient suspected of having mild active rheumatic infection They are also necessary in infants, mouth breathers and irrational, comatose and extremely ill patients *Infants and restless, irrational or comatose patients should be constantly watched while the thermometer is in the rectum, because of the danger of the instrument being broken*

Axillary Observation. The temperature of the axilla averages 1°F.–2°F lower than the mouth. It is obtained by placing the thermometer in the apex of the axilla and having the patient hold it there by pressing his arm firmly against his side Because it is much less accurate, this method should be used only when it is impossible to obtain oral or rectal readings.

Fever

Fever is present if the temperature is over 99°F by mouth or over 100°F. by rectum. A high fever which lasts more than a day or so and ceases abruptly (12–24 hours) is said to end by *crisis*, while one which subsides gradually ends by *lysis*.

Causes. Moderate rises of temperature are produced in the young by minor disturbances and are often less important than in adults

After childhood the vast majority of fevers are due to

1. Infection or inflammation
2. Toxemia without infection, as in cancer of the liver, lymphoma or other form of malignant disease, or reaction to some drug
3. Disturbance of heat-regulating mechanism, as in sunstroke, cerebral hem

it is not associated with a subsequent rise in temperature or other indication of an infectious or toxic state.

SINGLE CHILL is common at the onset of certain acute infections, notably lobar pneumonia, meningococcic, streptococcic and other pyogenic infections. Intravenous or intramuscular injection of whole blood, plasma, serum or some other agent may be followed by a chill.

RECURRENT CHILLS are most often due to malaria, general sepsis, and infection in the liver, bile ducts or upper urinary tract.

SUBNORMAL TEMPERATURE

Strictly speaking, a temperature below 98°F. by mouth is subnormal, but oral readings of 97°F. or even lower are not considered abnormal in a healthy person, especially after a sound sleep.

Persistently Low Temperature. This is most likely encountered in:

1. Low thyroid function.
2. Chronic wasting disease.

Acutely Developing Low Temperature. This is most commonly found in association with peripheral circulatory failure in such disturbances as

1. Trauma or exposure.
2. Extensive surgical operation or severe burn.
3. Sudden severe hemorrhage. Fever may appear later.
4. Acute severe illness such as diabetic coma, overwhelming systemic infection, myocardial infarction, acute intestinal obstruction or perforation, peritonitis, mesenteric thrombosis, anaphylaxis and severe drug poisoning.

SWEATING

Generalized Sweating. This occurs in a normal person with overheating, exertion and sometimes during sleep, especially a daytime nap. A sudden attack of nausea with or without vomiting may cause a normal person to break into a cold sweat; so will an emotional upset. Otherwise the most common causes are

1. Fever. Mild or moderate sweating is the rule during the course of most fevers, it becomes more pronounced during defervescence. Drenching sweats occur when the temperature falls abruptly.
2. Extreme prostration or weakness.
3. *Severe pain as in renal or biliary colic.*
4. Thyrotoxicosis.
5. Pregnancy.
6. Menopause, in association with hot flashes.
7. Peripheral circulatory collapse.
8. Extreme dyspnea, sometimes. Here it is the result of increased activity of the respiratory muscles.
9. Diaphoretic drugs.
10. Convalescence following operation, serious illness or childbirth.

Localized Sweating. Moist or actually wet hands, feet and axillae are common in emotionally unstable persons. They are also encountered in normal people

FIG 3 44 Chart of a case of lobar pneumonia showing recovery by crisis (Large circles represent fluid intake, large dots, urinary output)

causes in this country at the present time of obscure fever lasting three weeks or more are sepsis, including urinary tract infection, biliary infection and subacute bacterial endocarditis, tuberculosis, rheumatic infection, brucellosis, and malignant disease

CHILL

A true chill, manifested by a sensation of coldness, violent shivering and chattering teeth lasts from 5 to 15 minutes, rarely longer. It is promptly followed by a feeling of warmth and an abrupt rise in temperature The true chill must be distinguished from the chilliness or alternate hot and cold sensations present at the onset and during the course of any fever as well as from chilliness unassociated with disease The so-called nervous chill associated with an emotional crisis in a high strung person differs from the true chill in that

4

THE HEAD

THE CRANIUM

Size and Shape

Small Head. Microcephalus is most likely due to failure of the brain to develop normally It is also sometimes seen as a result of actual destruction of brain cells by hemorrhage or fetal encephalitis and occasionally may be caused by congenital syphilis Early closure of the sutures is a result, not a cause, of the small head

Large Head. The hydrocephalic head has a globular shape with a rounded vertex, the size of the cranium is often out of proportion to that of the face. The head of the achondroplastic dwarf is approximately of normal adult size but appears large because of the short limbs, prognathism and depressed nasal bridge are likely. In osteitis deformans *(Paget's disease)* the bone changes cause enlargement of the calvarium which in advanced cases is sufficient to produce disproportion between the size of the head as a whole and that of the face. The head and face are both enlarged in acromegaly.

Prominent Forehead. The frontal eminences and posterior parietal areas are prominent in rickets, causing a squarish outline which makes the head appear larger than normal but actual measurements usually show it to be within the normal range In congenital syphilis the frontal bosses are prominent and the breadth and height of the forehead exaggerated.

Nodules. Metastases of malignant disease occurring in or beneath the skin may be visible or palpable The most common are the bone lesions of multiple myeloma

Indentation. Following trauma a depressed area may be felt on the skull as a result of a depressed fracture. More often the palpable depression is due to central softening of a slightly raised hematoma produced by the injury

The Fontanels

The posterior fontanel closes about 6 weeks after birth. The larger anterior fontanel remains about the same size for the first year, then diminishes, closing about the sixteenth month The sutures ordinarily close toward the end of 6 months Closure is delayed in rickets, hydrocephalus, cretinism and sometimes in congenital syphilis.

during periods of stress. Profuse sweating of the feet, often malodorous and sometimes accompanied by nonodorous sweating of the hands, occurs in some normal persons without known cause. Otherwise the most common causes are:

1. Rickets. The head is the most likely site

2 Mediastinal tumor or aneurysm. Here the cause is sympathetic nerve irritation. The sweating is usually unilateral and most likely of the face and head, rarely of the trunk

3. Active rheumatoid arthritis Sweating of the hands and feet, usually cold, is common even when the joints of these parts are not involved.

4 Neurovascular disturbances of extremities In Raynaud's disease and following immersion or trench foot, the affected parts show excess sweating

Generalized Absence of Sweating. Diminution or absence of sweating with consequent dryness of the skin, occurs in heat stroke, myxedema, toxic doses of belladonna and its derivatives, and a number of skin disorders such as ichthyosis, scleroderma and severe psoriasis

Localized Absence of Sweating. Diminution or absence of sweating on the homolateral side of the face may occur with Horner's syndrome in cervical sympathetic paralysis due to a lesion such as aneurysm or tumor high in the thorax The upper extremity may also be affected. Interruption of a chain of sympathetic ganglia by injury or surgical operation will cause dryness and warmth of the skin over the denervated area.

scaliness and prominence of hair follicles; the hairs are broken off leaving loose stumps 2-3 mm in length

Favus of the scalp shows patches not unlike those of ringworm but distinguished by yellow cup shaped crusts (*scutula*). The hairs are brittle, broken off, often discolored and surrounded at their bases by scutula. In the well advanced case patches of baldness with scarring and atrophy of the scalp are present.

Graying of Hair. Although they are normal features of the aging process, graying and subsequent whitening may occur early, even before the thirtieth year in healthy persons, due to loss of function or death of the pigment cells of hair follicles The tendency often runs in families. Emotional strain may be a contributing factor. A single patch of gray hair is sometimes present without known cause.

Parasites. These should always be looked for. They are usually found in the less hygienic classes and inmates of crowded institutions, such as alms houses and orphan asylums. Persons with good hygiene may acquire them from contact: this is particularly true of nurses, teachers and others who work with the underprivileged. Often the parasites are not visible but one discovers ova (*nits*) attached to hair shafts. These are dirty-white, pear-shaped bodies with their ends pointing distally and are distinguished from dandruff scales by their characteristic shape and firm adherence to the hair If scratching has occurred, infection of the scalp and secondary cervical lymphnoditis may be found.

The scalp must be examined for such disorders as ringworm, favus, eczema, pyogenic infection, wens, warts and epidermoid carcinoma

Movements of Head

Constant tremor, shaking or nodding of the head occurs in old age, various neurologic disturbances, especially Parkinsonian syndrome and diseases affecting the cerebellum, sometimes in disturbed emotional states, and following excessive use of alcohol or drugs

THE FOREHEAD

Scar. This is usually due to trauma Multiple scars are frequently found in epileptics as a result of cuts sustained in falls

Eruption A rash may be a manifestation of an exanthematous disease or some local skin ailment The lesions of acne, smallpox, chickenpox, syphilis, drug eruptions and other exanthematous and dermatologic diseases can be readily confused, sometimes, to differentiate them, one must rely on history and other findings

Nodules. Local swellings or irregularities of surface beneath the skin may be the result of old trauma, syphilitic periosteitis or neoplastic disease

Supraorbital Swelling. Infection of a frontal sinus is the most likely cause The swelling may extend downward and involve the orbital tissues Local tenderness on percussion is a more reliable sign Puffy edema ascending upward toward the hairline as well as downward suggests osteomyelitis of the frontal bone secondary to sinus infection Swelling, tenderness, and thickening along



FIG 4 1 Paget's disease Note disproportion in size of cranium and face Temporal artery is dilated to meet the demand of diseased bone for increased blood (Courtesy Dr Fuller Albright)

In the normal infant bulging of the fontanel can be caused by any activity such as crying which increases intrathoracic and consequently intracranial pressure, in disease it is most likely due to hydrocephalus, intracranial hemorrhage, or meningitis

A depressed fontanel in an infant is found in wasting or dehydration from any cause

THE HAIR AND SCALP

Loss of Hair. Death of hair follicles can result from advancing years, infection or toxemia Premature baldness may be the result of a hereditary tendency to early death of hair follicles but seborrhea is almost always a contributing cause Diffuse and usually temporary loss of hair may occur following any severe or prolonged febrile illness, occasionally after emotional trauma Diffuse thinning and inability to take artificial curling are characteristic of myxedema. Irregular thinning creating a moth-eaten effect and appearing especially in the temporal and occipital regions is common in early syphilis A rachitic child often rubs the hair off the back of its head by constantly rolling on the pillow

ALOPECIA AREATA, the cause of which is unknown, is characterized by asymmetrically distributed, sharply-defined, variously shaped and sized patches of baldness disclosing smooth, shiny, unscarred scalp Exclamation-point hairs may be found, especially along the borders In some cases there is total loss of hair not only from the head but also from other regions where it normally grows.

TINEA CAPITIS is characterized by one or more circular patches of erythema,



FIG 4 2 Facial asymmetry due to seventh nerve paralysis Left eye is wide open, side of face droops, and nasolabial fold is flattened Medial deviation of left eye indicates sixth nerve paralysis

asis, starvation, pertussis, liver failure, obstruction to venous return and sometimes, especially in the morning, without known cause When combined with anemia, the puffy face has a peculiar waxy appearance; this is seen in many cases of nephritis

Cachexia. In the late stages of many wasting diseases, especially when there is pain or anemia, one may observe a characteristic haggard, shrunken appearance. This is common in advanced malignant disease and generalized arteriosclerosis. In late cirrhosis of the liver one sees also a yellowish tinge of skin and sclerae, and watery eyes.

Dehydration. Persistent vomiting, severe diarrhea or inadequate fluid intake as in neglected febrile illness gives the face a drawn, pinched, anxious look The nose is sharp, the eyes hollow, the cheeks sunken, the ears cold and shrunken, the skin dry and often showing a slightly cyanotic pallor This is essentially the *Hippocratic facies* first described by Hippocrates as occurring in severe acute illness In peripheral circulatory failure the picture may be similar except that the skin is clammy with cold sweat

Adenoids Especially in childhood, hypertrophied adenoid tissue or other cause of impaired nasal breathing such as chronic vasomotor rhinitis produces open mouth, pinched nose and somewhat stupid expression The palate is high and arched There is a history of mouth-breathing and snoring, frequent colds, and perhaps ear troubles.

Parkinsonian Syndrome. Progressive muscular rigidity results in gradual loss of the usual changes associated with mood, and eventually a totally mask-like expressionless countenance. The head and neck are usually inclined forward

the course of a temporal artery, sometimes with redness of overlying skin and arterial pulsation is seen in the syndrome, temporal arteritis Other important features are moderate fever of weeks' or months' duration, weakness, prostration, severe headache, pain in the eyes and throat, painful mastication, muscle aches, and loss of weight

Impaired Wrinkling. Diminution or absence of normal wrinkling of the forehead (*Joffroy's sign*) when the patient looks upward is seen in thyrotoxicosis In peripheral or nuclear seventh nerve paralysis the homolateral side of the forehead is smoother than the unaffected side and does not furrow on attempted frowning.

THE FACE AS A WHOLE

General Appearance

Even the untrained observer may gain an impression of a person's personality and temperament from his expression: the agitated appearance of the disturbed, the woebegone look of the depressed, the fatuous countenance of the mentally retarded, and the shifty eye of the unreliable

The trained physician has learned to associate certain appearances with certain diseased states. For example the anxious, flushed, dyspneic and perhaps cyanotic appearance of the patient with pneumonia provides an immediate clue to the diagnosis, it is quite in contrast to the dull, apathetic mien of the patient with typhoid fever, or the alert, shiny-eyed appearance of severe streptococcic septicemia Twitching and trembling may be a sign of agitation, it is also seen in the chronic alcoholic and the drug addict, especially when either is deprived of his usual dose Other disturbances in which the appearance of the patient is often striking are.

Asymmetry. This may be due to some minor defect such as loss of teeth on one side It is seen as a compensatory effect of chronic torticollis In peripheral (*Bell's palsy*) or nuclear seventh nerve paralysis there are homolateral loss of wrinkling of the forehead, widening of the palpebral fissure, inability to close the eye, flattening of the nasolabial fold, and drooping of the corner of the mouth which appears to be drawn toward the unaffected side Voluntary and involuntary movements of the affected muscles are impaired or absent In early paralysis due to a central lesion the picture is similar except that in all but the extreme cases the eye can be partially closed and the forehead wrinkled, later when contractures occur the mouth appears to be drawn toward the affected side (see Chap 36) In progressive facial hemiatrophy, which is presumably due to a lesion of the fifth nerve or its nucleus, asymmetry is caused by atrophy of soft tissues and perhaps retardation of bone growth on the affected side

Edema. When of slight degree, puffiness is more likely to be noticed by the patient or his friends than by the physician who is less familiar with his normal appearance, otherwise it is obvious Edema suggests nephritis or nephrosis but may occur in heart disease, diabetes, inflammatory disorder of the skin, trichini-



Fig 4 5 Leprosy

A Korean showing alopecia, loss of eyebrows, and diffuse infiltration and thickening of facial skin (Courtesy Dr David G Cogan.)

B Puerto Rican showing distinct lepromas creating leonine countenance and pendulous ear lobes (Courtesy Dr Thomas H Weller, Head, Dept of Tropical Health, Harvard University)

Rheumatic Heart Disease. A slightly cyanotic flush over the malar eminences and of the lips is seen in advanced rheumatic valvular disease with predominating mitral stenosis

Phthisis. In the early stage one may observe clear, delicate skin, fine hair, long eyelashes and wide pupils In advanced cases, pallor and febrile (*hectic*) flush appear

Leprosy. Alteration of the natural contours of the face by nodular and ridge-like thickening of skin and subcutaneous tissues causes so-called *leonine* appearance Skin and subcutaneous tissues may be shiny from atrophy. A staring appearance due to lid palsy or facial asymmetry may reflect total or partial involvement of a facial nerve Thinning of eyebrows occurs early Spotty alopecia of the scalp is common but is by no means pathognomonic, as it may be due to some other disturbance such as ringworm, which is also common in endemic areas A cord-like prominence in the neck due to thickening of the greater auricular nerve is often visible

Keratosis. Lesions are most likely to be seen on the forehead, nose, malar eminences or helix of the ear

Color

Significant changes in color are often most readily discernible on the face. They are likely to be overlooked in artificial light; adequate daylight is essen-

and in advanced stages the latter *is so rigid* that the head cannot be turned without rotating the body.

Chronic Alcoholism. There may be not only the traditional red nose but also—and oftener—a peculiar smoothed-out appearance, due probably to an extra but evenly distributed accumulation of subcutaneous fat. Many advanced alcoholics have a haggard look, coarsened and purplish skin, with telangiectases, especially on and around the nose, and reddish, watery eyes. Tremulousness is common *A ruddy complexion is normal with many persons, especially those who are plethoric or exposed to the elements.*

Acromegaly. The huge jaw and chin, large nose and ears, *prominent cheek* bones and supraorbital eminences are the striking features.

Thyrotoxicosis. The exophthalmos and quick movements of the eyes give the patient a startled expression.

Myxedema. In the well-established case the face is swollen, but in contrast to true edema, does not pit on pressure The skin is dry, sallow or yellowish, coarse in texture and lacking in elasticity. The tongue and lips are thick. The eyebrows, especially their outer thirds, are thinned. Because of the low metabolic rate, the patient has a somnolent appearance and slow responses which often lead to the erroneous impression of mental dullness. The swelling may be mistaken for that of nephritis

Myasthenia Gravis. The eyelids and jaw tend to droop; the face may have a blank expression because of weakness of the facial muscles



Fig 4 3



Fig 4 4

Fig 4 3 Thyrotoxicosis Note startled expression due to prominence of eyes. Fig 4 4 Myxedema.

Spasms of Face

Repeated, quick contractions of certain facial muscles (*tics*), such as winking-spasm, jerking of the corners of the mouth, or sniffing occur in the following:

1. Habit without disease. This type will sometimes spread through a school or institution.

2. Trigeminal nerve neuralgia. The spasm occurs with the attacks of pain and recurs in the same group of muscles.

3. Disturbances involving the central nervous system. By affecting particular sets of muscles, Parkinsonian syndrome, Sydenham's or Huntington's chorea, Friedreich's ataxia, birth injuries, and certain other neurologic disorders produce fairly characteristic patterns of movements of the face and head. Often these are associated with tremors or athetoid movements of the extremities. Spasms of the face are also seen in convulsive seizures.

4. Hysteria and disturbed emotional states

5 Mental disease.

6 Uremia, severe acute infectious disease and other toxic states

7. Alcoholism and drug addiction.

8. Tetany Tonic spasm of the facial muscles occurring continuously or intermittently may be one of the features of tetany (see Chap. 6) *Chvostek's sign*, a frequent but not pathognomonic indication of this disturbance is positive when contraction of the facial muscles on the homonomous side follows tapping the cheek along the course of the facial nerve. The contractions are best seen at the corner of the mouth or about the eye Chvostek's sign must not be confused with jaw-jerk elicited by tapping the masseter muscle.

9. Tetanus. Spasm of the facial muscles draws the corners of the mouth outward and the eyebrows upward, giving the effect of a mirthless grin (*risus sardonicus*) Strychnine poisoning produces a similar picture.

Tenderness

Light percussion of the face may reveal evidence of superficial or deep-seated inflammation The frontal region is commonly tender in acute frontal sinusitis, the malar eminence, in acute maxillary sinusitis Regional hyperesthesia is a frequent sign of trigeminal neuralgia and other disorders involving the fifth nerve or its branches.

THE EYES

Although the patient may have no symptoms referable to the eyes, they must always be examined with care because they so often provide clues to the diagnosis of systemic disease Only the more common local disturbances and those which are related to disease elsewhere will be discussed.

The Brows

Thinning of the eyebrows, especially their outer thirds, sometimes occurs in myxedema In secondary syphilis there is uneven loss of hair. Thickening of the

tial. In women, color variants may be concealed by cosmetics The most common changes seen in disease are:

Pallor. This is common in individuals who are poorly nourished, whose life is spent chiefly indoors or in the tropics or who are suffering from some chronic illness. It is usual in the anemic, but pallor does not necessarily signify anemia Pallor is pronounced in hemorrhage, peripheral circulatory collapse, and emotional shock.

Cyanosis. A bluish or purplish tint to the skin or mucous membranes results from impaired oxygenation or circulation of the blood. It is most commonly seen in

1. Heart disease, particularly mitral stenosis, cor pulmonale, certain congenital anomalies, and congestive failure from any cause.

2 Pulmonary disease, particularly emphysema, asthma, large pulmonary embolism, massive collapse, and severe pneumonia

3 Other disorders in which there is a disturbance of pulmonary ventilation, such as bronchial or tracheal obstruction, or of vascular flow, as in mediastinal tumor.

4 Peripheral circulatory failure from any cause Here the cyanosis is of a peculiar grayish or ashen hue.

5 Plethora, as in many cases of obesity, chronic alcoholism, polycythemia vera, and *pituitary basophilism*

6 Prolonged or excessive use of acetanilid, or certain other coal-tar preparations. These produce methemoglobinemia or sulphemoglobinemia which can sometimes be diagnosed by putting a *light behind the lobe of the ear or against* the inner wall of the cheek, and, with a small spectroscope held on the opposite surface, obtaining a characteristic spectrum If this test, in a suspicious case, is negative, a specimen of blood should be examined by the usual spectroscopic methods Sulfanilamide and some allied drugs, taken in therapeutic doses, produce cyanosis, and give the blood a spectroscopic band similar to that produced by methemoglobinemia or sulphemoglobinemia

Pigmentation. See Chap. 3

Jaundice. Deposition of bilirubin secondary to hyperbilirubinemia causes pale or deep yellow, orange, greenish yellow or sometimes almost green discoloration *of the sclerae, skin, and less strikingly, of the mucous membranes When mild, the color change may be observed* only in the sclerae, here it must be distinguished from the more patchy yellowness of subconjunctival fat Unless pronounced, jaundice is likely to be missed in artificial light In the darker *races, it is often overlooked in the skin but may be discernible* in the sclerae, the hard palate or, when they are compressed by a glass slide, in the lips When a case is equivocal or the hyperbilirubinemia is too slight to create visible tissue discoloration, *blood chemical determinations will clarify* the problem Carotinemia, quinacrine, dinitrophenol and certain other chemicals cause yellow color of skin or other tissues which may be mistaken for jaundice. The various types of the latter are *described subsequently* (*see Chap* 31)

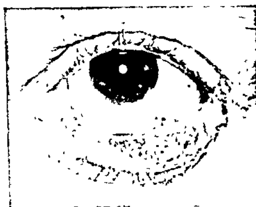




Fig 4 6 Fig 4 7

Fig 4 6 Ectropion of lower lid with mucopurulent discharge (Courtesy Dr David G Cogan)

Fig 4 7 Unilateral exophthalmus of five weeks' duration due to neuroblastoma of orbit in a boy age 4 (Dark line on nasal aspect of upper lid represents biopsy incision) (Courtesy Dr David G Cogan)

ing and palable crepitation as a result of seepage of air into the tissues following fracture of the nose or wall of a paranasal sinus The signs may be accentuated by blowing the nose.

Wide Palpebral Fissure. In thyrotoxicosis, retraction of the upper lids secondary to spasm of the levator muscles gives the patient a staring appearance (*Stellwag's sign*) Blinking is infrequent. Although almost always occurring equally on the two sides, this sign, as well as lid lag and globe-lag (see below), which are also encountered in thyrotoxicosis, may be highly predominant unilaterally.

Lid and Globe Lag These are best brought out by having the patient focus on a test object while it is slowly moved upward and downward in front of his eyes. When lid-lag is present the upper lid fails to keep pace with the globe as the gaze is lowered (*Von Graefe's sign*) In globe lag, the lid moves upward more quickly than the globe when the gaze is lifted.

Ptosis. Paralysis of the levator palpebrae, which is supplied by the third nerve, causes drooping and impaired elevation of the upper lid Affecting one or both eyes it may be congenital, existing as an isolated abnormality, or occur along with other manifestations of third nerve paralysis in encephalitis lethargica, meningitis, postdiphtheritic paralysis and idiopathic polyneuropathy Unilateral ptosis is one of the important signs of third nerve paralysis due to ruptured aneurysm of the intracranial segment of the internal carotid artery In botulism and myasthenia gravis, both lids droop, the weakness in the latter being primarily muscular. Slight drooping of the lid due to paralysis of Mueller's muscle which is part of the levator apparatus and is supplied by branches of the cervical sympathetic nerve is a feature of Horner's syndrome (see Pupils)

Impaired Closing. In Bell's palsy and other disorders affecting the seventh

skin and subcutaneous tissue beneath the outer third may be the first sign of leprosy.

The Lids

Edema. Swelling, especially of the lower lids, is frequently seen in normal persons when they arise in the morning, but soon disappears. It is sometimes due to lack of sufficient sleep or to alcoholic excess, but is also the first sign of edema of the face

In disease, edema of the lids is most commonly encountered in

1. Local lesions of the eye or lid
2. Inflammatory disturbances of the face, such as erysipelas, dermatitis venenata, dermatitis medicamentosa, and urticaria.
3. Measles and whooping cough.
4. Nephritis and nephrosis
5. Acute sinusitis
6. Anemia.
7. Diabetes.
8. Trichiniasis.
9. Thyrotoxicosis. In many cases it is associated with severe proptosis.
10. Myxedema.
11. Angioneurotic edema The swelling is usually localized to the region about one eye
12. Impairment of venous or lymphatic return as in cavernous sinus thrombosis or superior mediastinal syndrome.

Hordeolum. Otherwise known as *sty*, this is a circumscribed, acute inflammatory process occurring along the margin of the lid and characterized by pain, tenderness, redness, swelling and edema It develops into a pimple-like lesion with a yellow summit.

Chalazion. *Chronic inflammatory enlargement of one of the Meibomian* glands from obstruction of its duct causes gradual development of a non painful, circumscribed swelling of the lid It is hard and adherent to the tarsus but not to the skin Unless it becomes infected chalazion gives no acute symptoms or signs.

Ectropion. Eversion of the lid with chronic exposure of the eye is seen as a result of cicatricial contraction following injury, in chronic conjunctivitis or blepharitis, paralysis of the seventh nerve, persistent blepharospasm and sometimes in old age as a result of relaxation of skin and subcutaneous tissues Exposed conjunctiva is red and thickened, tearing is excessive and the cornea may become secondarily ulcerated.

Entropion. Rolling in of the lid margin is due to cicatricial changes from any cause or to spasm of the palpebral portion of the obicularis muscle, the latter occurring in elderly people, usually as a complication of the aging process The symptoms and signs are those of irritation of the conjunctiva and cornea

Subcutaneous Emphysema. The lids and surrounding tissues may show swell

tumor, cellulitis and hemorrhage. In leukemia, prominence of one or both globes is not uncommon. Arteriovenous aneurysm from injury behind the eye may cause pulsating exophthalmos

The Lacrimal Apparatus

Lacrimal Gland. This gland, situated in the upper lateral quadrant of the orbit just behind the upper orbital margin, is not normally visible or palpable. Inflammation or tumor causes localized swelling in the lateral portion of the upper fornix. The enlarged gland may be palpable through the lid; the latter may show slight localized bulging. *Diminished* tearing is most likely due to vitamin A deficiency or obstruction of the ducts by post traumatic scar tissue. *Excessive* tearing (*epiphora*) occurs with irritation due to could, wind, noxious fumes, foreign body and most inflammatory disturbances of the cornea Otherwise the most common cause is stenosis of the nasolacrimal duct or interference with free flow of tears higher up, such as occlusion by an eyelash, eversion of the lower tear point or maldevelopment of tear points

Mikulicz's disease is characterized by bilateral swelling of the lacrimal, parotid, and sometimes other salivary glands as a result of lymphoid infiltration. Similar swelling of these glands is observed in uveoparotid fever (usually regarded as a form of sarcoidisis) and may occur in other forms of sarcoidosis and in lymphoma. Such cases are referred to as *Mikulicz's syndrome*

Lacrimal Sac. This sac lies in a fossa of the lacrimal bone medial to the inner canthus and behind the internal tarsal ligament. Stenosis of the nasolacrimal duct may be congenital or result from chronic nasal infection, injury or syphilis. If uncomplicated, its only sign is excessive tearing but in longstanding cases, infection of the lacrimal sac (*dacryocystitis*) occurs This may be manifested only by slight localized swelling medial to and below the inner canthus. Sometimes pressure over the area may force a small amount of mucopurulent material back through one or both tear points. Recurrent attacks of acute inflammation with redness, tenderness, increased swelling and perhaps evidence of conjunctivitis may be superimposed

The Conjunctivas and Scleras

Conjunctivitis. Redness, congestion, and perhaps mucopurulent discharge and edema of the lids are evident The congestion is greater nearer the periphery; the hyperemic vessels appear superficial, somewhat wavy and bright red. In contrast, when the iris or cornea is involved, the congestion is greater near the cornea, the dilated vessels appear deeper, hence darker red, and are less tortuous Conjunctivitis occurs after undue exposure to bright light or fumes, as an independent infection, as a feature of any severe intra-ocular inflammation, and in acute coryza, hayfever, measles, pertussis, trichiniasis, typhus fever, yellow fever, tularemia and other systemic disorders In thyrotoxicosis, especially cases of so-called malignant exophthalmos, and in seventh nerve disorders, conjunctivitis and inflammation of other parts of the eye may occur as a result of drying due to incomplete closure of the lids and infrequent blinking

nerve, paresis of the *orbicularis oculi muscle is indicated* by weakness and lagging of the lids when the patient attempts to shut the eye. When paralysis is *complete the eye remains open.*

Infraorbital Darkening. Dark circles under the eyes are normal in some people; in others they indicate fatigue They have little significance.

The Eyeballs

Intraocular Pressure. *Intraocular pressure can be roughly estimated by palpating* the eyeballs through the closed lids, with the patient's head resting against *a firm support* Whenever *determination* of tension is important the tonometer must be used.

Diminished Tension. In dehydration from any cause the eyeballs feel soft; with improvement, normal tension is restored. This is especially characteristic of diabetic coma Hypotony occurs as a permanent sequela of serious damage to the ciliary body, as in severe iridocyclitis

Increased Tension In glaucoma, the eyeball feels hard An *acute* attack is *marked by rapid appearance of pain, often severe and extending over the head,* edema of the lids, lacrimation, congestion of conjunctivae, edema and clouding *of cornea, dilatation of pupil,* and marked reduction of vision *If the retina is* not obscured by corneal clouding, engorgement and pulsation of its vessels will be seen Nausea and vomiting are frequent, they may lead to the erroneous diagnosis of an acute gastric upset. In *chronic* glaucoma, pain and conjunctivo-corneal changes are less striking than in an acute episode, loss of vision is gradual and, except in the late stages, affects only the peripheral fields When the process is well-established, cupping of the optic disc is seen

Position. The position of the globe in the orbit can be roughly determined by noting its relation to the eyelids in their normally opened state If the eye-*ball is prominent, the sclera is visible above and below the cornea, if retracted,* more of the cornea is covered than is normal These criteria are not entirely dependable. Retraction of the lids may cause the illusion of *prominence,* the globe may appear sunken if the upper lid droops In doubtful cases, one must use the exophthalmometer to measure how far the plane of the corneal apex extends beyond the lateral rim of the orbit In most normal persons the distance is 15–20 mm

Enopthalmos In dehydration the eyeballs are sunken from loss of fluid, in wasting diseases and sometimes in old age from loss of intra-orbital fat. Enophthalmos, unilateral or bilateral, is sometimes congenital or may result from contraction of scar tissue following orbital infarct, injury or infection In *Horner's syndrome mild enophthalmos may* occur but as a rule the illusion of enophthalmos is created by drooping of the lid

Exophthalmos (Proptosis) Bilateral prominence of the eyeballs is seen in some normal persons; it is often a familial characteristic It occurs in many *myopic* persons due to elongation *of the globe Developing after childhood,* bilateral, or rarely predominantly unilateral, exophthalmos is likely due to thyrotoxicosis *Unilateral* proptosis, sometimes actual *bulging, occurs in orbital*

THE CORNEAS

Arcus Senilis. This is a grayish-white arc partially or totally circling the periphery of the cornea, but never extending to the limbus. Due to lipoid infiltration, it is seen frequently in old age, occasionally in younger persons. It is usually of no special clinical significance, but may be an indication of hypercholesteremia.

Band Keratopathy. Subepithelial calcification, most marked in the palpebral fissure of the cornea near its periphery and fading toward the center, occurs in various degenerative disturbances of the eye. It is also seen in otherwise normal eyes in hypercalcemia due to such causes as hyperparathyroidism, sarcoidosis, vitamin D intoxication and uremia. Although best observed with the slit lamp it can frequently be detected by the naked eye with good illumination. It is recognized by grayish opacity in the paralimbal region. It must not be confused with arcus senilis, which is not confined to the palpebral area and with the slit lamp can be seen to involve the entire thickness of the cornea.

Interstital Keratitis. An important sign of congenital syphilis, this usually occurs in children and young adults. It is manifested by diffuse clouding of the cornea with moderate circumcorneal injection, the evident presence of blood vessels extending into the cornea, and, in the acute stages, severe photophobia. In the later stages diffuse or localized areas of deeper clouding are present. The diagnosis is confirmed by other signs of congenital syphilis, especially the so-called Hutchinsonian teeth, the saddle nose and positive serologic reaction.

Corneal Ulceration. ACUTE ulceration is marked by redness predominating in the circumcorneal area and cloudiness of the cornea. The color of the congested zone is darker red than in conjunctivitis and the vessels which seem to radiate



FIG 48   FIG 49

FIG 48 Arcus senilis. Note that clouding does not extend to limbus. (Courtesy Dr David G Cogan.)

FIG 49 Band keratopathy, left eye, due to hypercalcemia. Calcium deposits have clouded the cornea and obscured iris details in paralimbal region. Process most pronounced on nasal side but also evident on temporal side. (Courtesy Dr David G Cogan.)

Conjunctivitis may result from large doses of potassium iodide, arsenic, and other drugs; in susceptible persons it follows small doses If the cornea is anesthetic as a result of damage to the orbital branch of the fifth nerve, presence of a foreign body of which the patient is unaware may cause serious infection of the conjunctiva and cornea Gonorrhea is suggested by a profuse mucopurulent discharge, *diphtheria by a membrane attached usually to the conjunctiva of the lower lid which, if removed, exposes a raw ulcerated surface*

ACUTE CATARRHAL CONJUNCTIVITIS Popularly known as *pinkeye*, this is characterized by intense redness, serous or mucopurulent secretion and perhaps edema of the lids It is caused by a number of different organisms, most commonly the pneumococcus, and is likely to occur in epidemics, especially in schools and institutions

CHRONIC CATARRHAL CONJUNCTIVITIS. Due to a variety of causes such as low-grade infection, an irritant or some allergen, this is indicated by moderate redness, thickening and, usually, tearing In chronic alcoholism the eyes may be *watery and hyperemic.*

TRACHOMA This serious, contagious and usually chronic disease occurs most commonly where unhygienic living conditions prevail At onset the picture is similar to acute catarrhal conjunctivitis, but after a week or more, the palpebral conjunctiva becomes thickened, velvety and studded with beefy-red follicles which gradually increase in size and give the lid a granular appearance After several months, these follicles are gradually replaced by scar tissue which appears as *gray or whitish lines Entropion or ectropion and involvement of the* cornea by ulceration and opacities are common complications

Petechiae. When subacute bacterial endocarditis is suspected, the conjunctivas should be inspected from day to day for the appearnace of one or more small petechial hemorrhages which might be diagnostically important

Discolored Sclera. In conjunctival hemorrhage, the sclera appears *red* due to deposit of blood in overlying conjunctiva Especially in the darker races, the *yellow* color of jaundice *may be recognized in the sclera when it is not sufficiently* deep to be seen in the skin It can readily be overlooked in artificial light It must not be confused with subconjunctival fat, which appears in spots and patches, not with even distribution Prolonged use of quinacrine will cause yellowish discoloration which is easy to mistake for jaundice. In the former, the change is most marked around the limbus and fades toward the fornices, in jaundice, the reverse is true. If quinacrine is the cause, the palate and nail beds may be grayish-blue *Occasionally, prolonged use of silver salts in the treatment*

always spared Alkaptonuria shows a characteristic spotty or linear *brown* discoloration of the episcleral tissues. In idiopathic hypochromic anemia the scleras may appear *pearly-white* or have a *bluish* tinge, in contrast to pernicious anemia, in which they are yellowish The scleras are *blue* in osteogenesis imperfecta, a rare *familial disease.*

zoster virus occurs in association with vesicular dermatitis over the area innervated by the first division of the trigeminal nerve. It is usually associated with marked pain and some degree of uveitis In contrast to herpes simplex, the ulcer is not dendriform.

The Irises

Iritis. Pain, lacrimation and disturbance of vision are usually present. One observes swelling, loss of luster and discoloration of the iris, a small and not easily dilatable pupil, and circumcorneal injection resembling that of keratitis. Iritis suggests a focus of infection in a tonsil, tooth, sinus or other structure, or some systemic disease such as syphilis, tuberculosis, diabetes, sarcoidosis, rheumatoid arthritis or chronic brucellosis. It should not be confused, as it often is, with acute glaucoma, of which the distinguishing features are severe pain, greater impairment of vision, dilated pupil and increased intraocular tension

Sarcoma. The iris shows an elevated area of pigmentation which grows fairly rapidly and may partially obliterate the pupil

The Pupils

Examination of the pupils is of special importance in the diagnosis of certain disorders affecting the nervous system and in the localization of intracranial lesions. Abnormalities of pupillary size, shape, reaction to light and distance can, however, result from injury or local disease of the iris or cornea and do not necessarily reflect a remote disturbance.

To avoid consensual reaction the pupillary responses of each eye must be tested with the opposite eye covered. The *light* reflex is best obtained in a dark room; to exclude the accommodation reflex the patient must focus on a distant object The normal response is immediate contraction of the pupil when a bright light is directed toward the eye The *accommodation* reflex is obtained by having the patient look first at a distant object and then at one held within reading distance. To exclude the light reflex, the degree of illumination must be constant The normal response is contraction for near focus, dilatation for distance

Dilated Pupils. Common causes are blindness or extremely deficient vision, strong emotional reaction, many fevers, comatose states, glaucoma, third nerve paralysis and local or internal administration of mydriatic drugs such as cocaine, atropine and epinephrine In subdural hematoma, the pupil may be dilated on the affected side

Constricted Pupils. Common in old age and in photophobia, constricted pupils are seen also following the use of morphine, pilocarpine, or other miotic drug Contraction, with irregular outline and sluggish or absent reactions, is often seen in iritis as a result of adhesions to the lens (*posterior synechiae*), and in neurosyphilis. A lesion involving the cervical sympathetic chain or the sympathetic pathways in the brain stem or spinal cord may produce a constricted pupil, together [illegible]
illusion of enop



FIG 4 10. Opacity lower part of cornea due to purulent infiltrate beneath a corneal ulcer (Courtesy Dr David G Cogan)

outward from the cornea appear deeper and less tortous. Photophobia is more pronounced than in conjunctivitis

CHRONIC ulceration presents a picture not unlike interstitial keratitis, except that the clouding is confined to localized areas rather than being diffuse and is accompanied by superficial loss of substance Inflammation of the cornea, as well as of the lids and other ocular structures, may occur in various dermatologic, infectious and other systemic diseases

In any inflamed eye, the cornea should be carefully searched for a foreign body.

Xerophthalmia. Resulting from avitaminosis A, this is characterized by a dry, lusterless, shrunken conjunctiva upon which are imposed minute, cheesy flakes Sometimes the cornea has a glassy appearance; in severe cases it is opaque Ulceration may occur Nightblindness (*nyctalopia*) is often the earliest symptom of vitamin A deficiency.

Superficial Vascularization. This is said to occur in riboflavin deficiency It is accompanied by photophobia, lacrimation, and sometimes dimness of vision

Phlyctenular Keratoconjunctivitis One or more small, gray or yellowish *elevations or nodules appear on some part of the conjunctiva or cornea* The lesions are usually surrounded by zones of redness, whereas the remainder of the conjunctiva is fairly normal Ulceration may occur The phlyctenules usually come in crops, lasting a few days to a week or more Children, especially those who are debilitated, are most often affected A relation between this disease and tuberculosis is thought to exist but has never been proved

Rosacea Keratitis. Occurring as a complication of acne rosacea, this is characterized by small nodules or marginal infiltrates of the cornea. Scar formation interfering with vision may develop.

Herpetic Keratitis. The usual form, caused by the herpes simplex virus, is characterized by a branched ulcer (*dendritic keratitis*) Since the cornea becomes anesthetic, pain is usually slight. A less common variety caused by the herpes

zoster virus occurs in association with vesicular dermatitis over the area innervated by the first division of the trigeminal nerve. It is usually associated with marked pain and some degree of uveitis. In contrast to herpes simplex, the ulcer is not dendriform.

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Sarcoma. The iris shows an elevated area of pigmentation which grows fairly rapidly and may partially obliterate the pupil.

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on the homolateral side of the face. This combination of signs, known as Horner's syndrome, may be observed with aortic aneurysm or apical intrathoracic tumor (*peripheral involvement*), syringomyelia (*spinal involvement*), or with disease at a higher level such as infarct of the medulla.

Unequal Pupils. Inequality of the pupils results from any disorder which dilates or constricts one but not the other. Iritis, neurosyphilis, and sympathetic paralysis, in particular, must be kept in mind. The unequal pupils of neurosyphilis are usually of the Argyll-Robertson type (see below); the others are not. Difference in size is also seen in tuberculous meningitis, less often in other forms of acute meningitis, and in other intracranial diseases. Occasionally it occurs without known cause.

Argyll-Robertson Pupil. The pupil reacts to distance but not to light and, as originally described, is also small, but the term is now generally used to denote any pupil showing these responses, irrespective of its size. The true small Argyll-Robertson pupil is an important finding in neurosyphilis, notably tabes dorsalis and dementia paralytica, and is seldom observed in any other situation. In either of these diseases, however, one may find a large pupil showing the characteristic Argyll-Robertson responses. *Diseases affecting the midbrain usually show normal-sized or large pupils which fail to react either to light or distance*, rarely a typical Argyll-Robertson pupil. *Following encephalitis lethargica the opposite responses are frequently observed*—the pupils respond to light but do not accommodate for distance.

Adie's syndrome, seen usually in young women, is of unknown etiology. One or both pupils are moderately dilated and show absent or greatly diminished direct and consensual response to light when the test is performed in the usual manner. However, they will dilate after an hour or more in the dark and, if then exposed to a bright light, will slowly contract. The accommodation reflex is slow but the range of dilatation and contraction may be greater than for light. In contrast to the Argyll-Robertson pupil, the response to certain miotics is greater than normal. *These pupillary variants are often associated with absence of one or more tendon reflexes of the lower extremities and occasionally of the upper extremities; if both ankle jerks are present, loss of other reflexes is rarely observed.* Failure to recognize this syndrome often leads to an incorrect diagnosis of tabes dorsalis.

The Lenses

Cataract. This is an opacity of the lens usually coming on in late life (*senile cataract*) but may be present at birth (*congenital cataract*). In the early stages it causes blurring of vision and can be detected only by ophthalmoscopy which will reveal a black silhouette against the fundus reflex. Later it produces severe impairment of vision and a white pupillary reflex, readily seen with the

atopy.

Ocular Movements

The third, fourth and sixth cranial nerves control the movements of the eyeball. In a normal person the image of an object is focused on the macula of each eye and the two images are fused into one, giving binocular single vision. When one eye deviates, the two images are not fused, so that diplopia will result unless the patient learns to disregard the image in the deviating eye.

Strabismus (Squint). If one eye is turned outward, squint is called *divergent* or *external*, if inward, *convergent* or *internal*.

Paralytic squint is marked by total or partial paralysis of one or more of the extrinsic ocular muscles due to a lesion somewhere in the nerve pathway, either central or peripheral. With third nerve paralysis, the eye cannot be moved inward, upward or downward, with sixth nerve paralysis, laterally; with fourth nerve paralysis, downward and outward. Deviation may or may not be apparent when the gaze is directed straight ahead but is evident when the patient, with his head stationary, directs his gaze in the direction requiring use of the affected muscle. Abnormal position of the head may be assumed in an attempt to compensate for the diplopia. In adults, rarely in children, with only a slight degree of muscle weakness, diplopia may be complained of but the squint may be detectable only by vision tests.

Paralytic squint may exist at birth from trauma or from congenital aplasia of an ocular motor nucleus. When acquired, it may be due to trauma, neurosyphilis, tuberculous meningitis, encephalitis, multiple sclerosis, diphtheritic paralysis, myasthenia gravis, a lesion of the brain stem, or other intracranial lesion.

Concomitant squint shows no true paralysis. There is no limitation of movement of either eye as the gaze shifts, the degree of deviation of the affected eye with respect to the normal eye remains quite constant with changes in direction of the gaze. This disorder is often hereditary but may not be noted until two or three years after birth. It is usually due to a high degree of hypermetropia or to poor vision in one eye. If present since infancy or early childhood, it is probably not associated with any systemic disorder. Failure of the eyes to perform normal conjugate movements with consequent double vision may occur in alcoholism, various toxic states and extreme weakness from any cause.

Nystagmus. Short, rapid, involuntary oscillation of both eyeballs may be congenital but is often acquired. It is most likely in the horizontal plane, sometimes around the anteroposterior axis, rarely vertical. Usually similar in the two eyes, the oscillations may be apparent or can be brought out only by having the patient look as far as possible first to either side, then upward or downward, attempting in each instance to hold his gaze fixed.

Congenital nystagmus is sometimes due to greatly diminished vision from such disturbances as corneal opacities, intraocular disease and marked refractive errors. It is common in albinism.

Acquired nystagmus is common in severely deficient vision and sometimes develops in miners, presumably as a result of poor illumination. Of more

importance is its occurrence in multiple sclerosis, cerebellar or medullary tumor, infarct or abscess, *syringomyelia, and disease of the labyrinth.* When due to a unilateral lesion of the cerebellum or brain stem, the nystagmus is most pronounced on directing the gaze to the homolateral side; in vestibular disease, to the opposite side When vertical oscillations are detected, one can be virtually certain that a central nervous system disorder exists.

Nystagmus must be distinguished from movements due to weakness of lateral gaze in a lesion such as thrombosis of the opposite cerebral hemisphere; here *muscular weakness prevents the patient from maintaining full lateral gaze* so that the eyes tend to drift back toward the primary position.

Moebius's Sign. The eyes are unable to converge properly when the gaze is fixed on a near object This is observed in thyrotoxicosis and postencephalitic Parkinsonian syndrome

Visual Acuity

Determination of the acuity of vision is of value in detecting a number of local ocular and neurologic defects and may call attention to those associated with some systemic disturbance The Snellen and Jaeger letter charts provide a simple method of testing, *respectively, distant and near vision* Each eye must be examined separately

Distant Vision. This should be tested first The Snellen chart presents a series of letters scaled in size and thickness so as to be normally visible at distances of 20, 30, 40, 50, 70, 100 and 200 feet. Tests should be performed with the patient 20 feet away because at this distance, rays of light from an object are essentially parallel and no accommodation is necessary If the patient reads from 20 feet *the letter normally visible at that distance, his visual acuity is normal*, or 20/20, if he can see only the letter which should be visible at 30 feet, his acuity is 20/30, and so on down the scale to 20/200. The person who cannot see at 20 feet the letter normally visible at 200 feet moves gradually toward the chart until this letter does become visible. His vision is then recorded as a fraction of 200, for example, one who can see the letter from 8 feet is said to have 8/200 vision. When the largest letter is not visible at any distance, he is tested for ability to *count fingers, perceive hand movements, determine light projection* or perceive light at all, and the results are so recorded.

Near Vision. The Jaeger card presents lines of various-sized printer's types [illegible] being No. 1. The card is held at the normal [illegible] rded. [illegible] yopia will tend to move the card further away; the one with myopia will move it *closer Most normal persons over 45 have lost accommodation* and cannot read ordinary type at a comfortable distance.

Reduction of Visual Acuity. Common disturbances causing impairment of vision are:

1 Error in refraction. myopia, hypermetropia or astigmatism.
2. Opacities in lens, cornea or vitreous.
3 Hemorrhage into retina or vitreous

4. Vascular disturbance such as embolus of the retinal artery, thrombosis of retinal vein or transient arterial disorder (presumably spasm) likely to be encountered in arteriolosclerosis and uremia.

5. New growth of the choroid or retina.

6 Retinal disease or detachment.

7 Glaucoma.

8 Iritis.

9 Affections of the optic nerve: optic atrophy, optic neuritis, retrobulbar neuritis, chronic glaucoma or toxic amblyopia

Color Perception. The *Ishihara* test is the most widely used The patient looks in turn at a number of colored cards. Each card has a background of colored dots into which is blended a figure composed of dots of a color likely to be confused with that of the background. The person with normal color perception will see the superimposed figure; the color-blind person will not. Examination for color perception is of importance in railroad, flying and seagoing personnel and others required to recognize signal lights. Approximately 4 per cent of males and 0 3 per cent of females are congenitally color blind, most commonly to red and green. Loss of color perception may develop in diseases of the retina and optic nerve.

Fields of Vision

When some disorder such as optic atrophy or an intracranial lesion is suspected, one must carefully plot the visual field of each eye using the perimeter for the peripheral fields and the tangent screen for the central fields

If the patient's condition makes these tests impracticable or a rough estimate is sufficient, one may use the *confrontation method* performed as follows: The examiner, with his hand covering one eye, sits facing the patient at a distance



Fig 4 11 Confrontation method of testing visual field

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Near Vision. The Jaeger card presents lines of various-sized printer's types numbered in sequence, the smallest being No 1 The card is held at the normal reading distance of 40 cm and the smallest type the patient can read is recorded If vision is normal, the No 1 type is clearly visible. A person with presbyopia will tend to move the card further away, the one with myopia will move it closer Most normal persons over 45 have lost accommodation and cannot read ordinary type at a comfortable distance.

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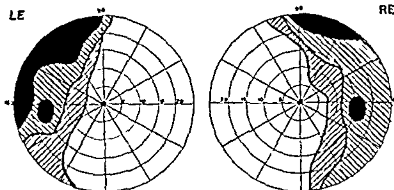


FIG 4 13 Fields of vision Incomplete bitemporal hemianopsia due to pituitary adenoma (Courtesy Dr David G Cogan)

Every physician should be familiar with the appearance of the normal fundus and the changes encountered in the more common systemic disorders.

Direct examination with the electric ophthalmoscope is best A dark room is essential If the pupils are small, dilatation with a mydriatic may be necessary; in this event examination of the pupillary reflexes should be completed beforehand *Mydriatics should never be used in any case in which there is the slightest suspicion of glaucoma* suggested by a history of blurred vision or halo images before the eye, detection of a shallow anterior chamber or, on palpation of the globe, the impression of more than normal hardness The same applies when there is reason to suspect increasing intracranial pressure, a situation in which it is important to watch for change in size of the pupils

The patient is instructed to look straight ahead and, in order to eliminate the pupillary contraction of accommodation, to fix his gaze on some distant object The examiner, with his ophthalmoscope adjusted for use of the 4+ lens, holds the instrument close to his own eye and directs the light through the patient's pupil from a distance of 10–15 cm In the normal eye, a homogeneous orange-red color (*red* or *fundus* reflex) is obtained Clouding in the media (cornea, aqueous, lens or vitreous) will give a reflex varying from dull orange to gray or black, circumscribed opacities will appear as dark spots on the red background.

With the ophthalmoscope still close to his own eye but adjusted for use of the 0 lens he now brings it to within 1–3 cm. of the patient's' eye Unless clear vision is interfered with by medial opacities, the nerve head, vessels and retina are clearly visible, provided the examiner is emmetropic or wearing proper glasses and there is no appreciable refractive error in the patient's eye If they are not well seen, the graduated minus and plus lenses are tried in turn until the one which provides the clearest view of the fundus is found. The strength of the lens required provides a rough index of the error of refraction plus lenses indicate far-sightedness, minus lenses, near-sightedness Obviously, the stronger the lens, the greater the refractive error.

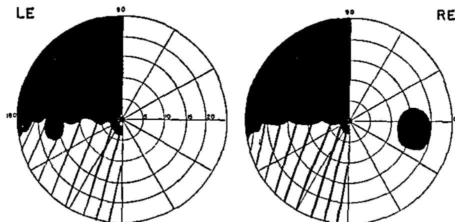


FIG 4 12 Fields of vision Homonymous left field defects due to tumor in right temporal lobe In this reproduction and in Fig 4 13, diagonal lines represent partial loss of vision, black areas, total loss, isolated black oval areas, normal blind spots (Courtesy Dr David G Cogan)

of approximately 2 feet The patient, with the opposite eye covered, fixes his gaze on the uncovered eye of the examiner The latter then moves his finger or, preferably, a small, white object inward from all sectors of the periphery of the intervening field in the plane midway between patient and examiner If the patient's field of vision is normal, he will see the test object at approximately the same time as does the examiner If his field is defective he will not see the *test object in the zone corresponding to the part of the field affected The usual* types of field defect are.

Homonymous Hemianopsia. Reduction of vision affecting the same side of both fields indicates a lesion in the opposite side of the brain

Bitemporal Hemianopsia. Reduction of the two fields on their temporal sides usually indicates a lesion affecting the optic chiasm, most commonly pituitary adenoma, suprasellar cyst or meningioma

Predominant Loss of Central Vision. General impairment of vision, but with greater loss of acuity centrally than peripherally, occurs with optic neuritis, retrobulbar neuritis, toxic amblyopia, degenerative lesions of the macula and obstruction of the central retinal artery

Predominant Loss of Peripheral Vision. The loss may be concentric or affect a sector of the field The most common causes are optic atrophy, chronic glaucoma, retinitis pigmentosa and retinal detachment In hysterical amblyopia, *the field of vision is contracted concentrically and may be of the tubular or* "gunbarrel" variety, that is, the field is not only constricted, but does not increase in size as it should, when the test object is moved further away from the eye.

THE FUNDI

Although the finer points of ophthalmoscopy must be left to the specialist, examination of the fundus should be part of a complete physical examination

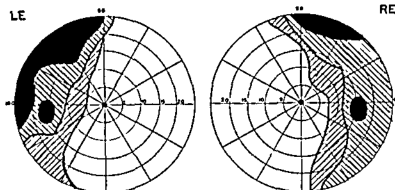


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First the nerve head is studied, then the vessels at the nervehead and along their courses toward the periphery, and finally all observable areas of the retina The regions not in the immediate vicinity of the disc can be brought into view by having the patient slightly shift his gaze in various directions without moving his head

The normal fundus shows considerable variation from person to person; experience is required to distinguish between physiologic differences and lesser degrees of pathologic change.

The degree of elevation or depression of the nerve head may be roughly measured in terms of diopters, by noting the difference between the strength of the lens which gives the clearest sight of the nerve head and that which gives the clearest sight of adjacent retina Unless there is choking or considerable depression of the disk, both the nerve head and retina are best seen with the same lens Opacities in the cornea, vitreous body, or lens are studied with the high plus lenses of the ophthalmoscope

Optic Disc

The nerve head is circular to oval and has a sharply defined margin which, especially on the temporal side, may be bordered by a ring or crescent of pigment It is light pink, perhaps slightly darker on the nasal half; just to the temporal side of its center the color is pale, sometimes white and the surface may be slightly depressed *(physiologic cup)* Medially to this area emerge the central artery and vein, dividing into their superior and inferior branches,

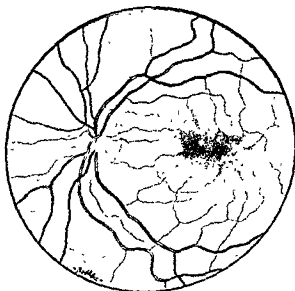


FIG 4 14 Normal fundus, left eye From May's *Manual of Diseases of the Eye*, 21st edition, The Williams & Wilkins Co., Baltimore 2, Md , 1953 Reproduced with permission of Dr Charles A Perera

which cross the disc and extend into the surrounding retina. Further major branching usually occurs before the vessels reach the disc margin, and smaller twigs are always seen on its surface. Common variations from the normal are:

Redness. Hyperemia is found in eye strain from such causes as hyperopia, astigmatism, exposure to glare and in papillitis and papilledema.

Pallor. In primary optic atrophy, degeneration of the nerve causes the disc to fade from the normal pink to milky-white; in secondary optic atrophy, glial proliferation makes it grayish-white

Elevation. The earliest manifestation is blurring of the disc margins; next, because of edema, the actual outline of the structure becomes less distinct. In advanced cases, the nerve head becomes definitely raised above the level of the surrounding retina, a stronger lens is needed to bring it into focus than is required for adjacent retina. The disc margins are obscure or obliterated and perhaps surrounded by a zone of small hemorrhages. To distinguish pathologic from slight normal physiologic blurring it may be necessary to plot the size of the blind spot with the aid of the tangent screen and test objects. In pathologic blurring the blind spot is larger than normal.

Depression. Varying from flattening to definite cupping, depression is seen in optic atrophy, primary or secondary, and frequently in glaucoma. To focus on the disc one will require a more concave lens than for the retina.

Retinal Vessels

The term, *arterioles*, is used for all of the arterial vessels with the exception of the central artery and its major branches They appear as red, slightly wavy lines, emerging from the center of the disc, crossing it and extending in various directions over the retina, branching as they go They are normally narrower, straighter and brighter red than the veins, along its axis each shows a thin, bright stripe (*light reflex*).

In the simple type of arteriolosclerosis some branches of the arterioles appear narrower at certain points, with perhaps a widened light reflex and some degree of arteriolovenous nicking or compression (see below) In advanced cases, one observes more pronounced patchy changes, greater arteriolovenous nicking, and tortuosity much greater than the slight waviness which is regarded as normal. Thickening may be sufficient to give some of the vessels a coppery color (*copper-wire arterioles*), others may appear as thin white lines (*silver-wire arterioles*)

In hypertension, there is generalized constriction of the arterioles which is said to vary from time to time The light reflex is thinner. Depending on the degree of sclerotic change, arteriolovenous nicking may or may not be present.

The *veins* which follow the same courses as the arterioles normally are darker red, wider, somewhat more tortuous and show a faint or no light reflex They may pulsate When depressed by a crossing arteriole which is sclerotic, the vein seems to narrow or disappear entirely at the point of crossing (*arteriolovenous nicking* or *compression*) and may be larger distally than proximal to the intersection. Venous dilatation and tortuosity are seen in choked disc, extreme hypertension and, distal to the lesion, in thrombosis of a retinal vein

Retina

Although the retina itself is transparent, its pigment epitheleal layer and the background of the choroid give it, in blondes, a bright orange, finely granular, and in brunettes, a deeper, brick-red appearance. In Negroes it may look slate-gray. When retinal pigment is scanty, the choroidal vessels may be visible as anastomosing channels but are by no means as sharply defined as the retinal vessels If the choroidal pigment is abundant it appears as dark patches between the vascular spaces of the choroid giving the retina a tessellated appearance. About two disc-diameters laterally to the disc, one can observe the *macula* This is somewhat darker than the surrounding retina; the vessels converge toward it, but except for a few barely visible twigs, do not appear to enter it The *fovea centralis* is represented by a small circle, slightly darker red and within its center, a point of light. Common deviations from the normal are

Edema. In the early stages, edema causes the retina to appear thicker and more opaque than normal, shimmering, irregular light reflexes may be noted Later, the color is milky, the vessels seem elevated and show diminution of their light reflexes

Hemorrhages. These appear as homogeneously red areas of discoloration, when large and dense they have slaty-blue centers They vary in size, depending on the cause, and are more profuse centrally than peripherally Occurring in the superficial layers of the retina, they are flame-shaped, in the deeper layers, round, oval or irregular. They may disappear completely or end in small yellow or whitish areas of scarring.

Exudates. Soft, ill-defined, white or slightly yellowish patches, discrete or confluent, of varying size (*cottonwool patches*) or small discrete white dots are observed. They tend to follow the courses of the retinal vessels and are likely to be distributed radially around the macula (*macular star*)

Common Disturbances of Fundi

Papilledema (Choked Disc). This term is used when swelling of the nerve head is attributable to increased intracranial pressure. The most common causes are brain tumor, abscess, meningitis, some cases of intracranial hemorrhage, delayed venous return as in cavernous sinus thrombosis or orbital tumor, and the late stages of retinopathy resulting from the vascular changes which occur in hypertension, nephritis, toxemia of pregnancy, and sometimes in terminal leukemia. At first the physiologic cup disappears and the disc margins become blurred and irregular. Later the nerve head is raised, the retinal veins are distended and tortuous, and hemorrhages about the disc margins are likely Optic atrophy may be the end result

Optic Neuritis Optic neuritis is encountered in encephalitis, multiple sclerosis, neurosyphilis, chemical poisoning, especially lead and methyl alcohol, and, rarely, in acute infectious diseases, notably mumps The picture is comparable to that of early papilledema but the nerve head is less swollen Here, too, secondary optic atrophy may develop Reduction of vision is more pro-

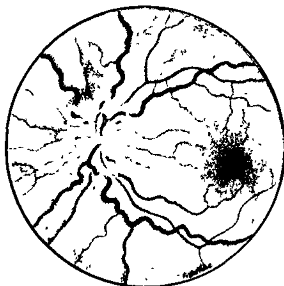


Fig 4 15 Papilledema Blurred margin of disc and dilatation and tortuosity of retinal veins From May's *Manual of Diseases of the Eye*, 21st edition, The Williams & Wilkins Co, Baltimore 2, Md, 1953 Reproduced with permission of Dr Charles A Perera.

nounced than in papilledema except when the latter has advanced to the stage of optic atrophy. In retrobulbar neuritis, similar inflammatory changes affect the retro-ocular portion of the optic nerve, producing serious visual impairment, but the disc appears normal unless optic atrophy occurs. Toxic amblyopia resulting from prolonged use of alcohol and tobacco, less often caused by other poisons such as carbon monoxide, benzol, carbon tetrachloride, carbon bisulphide and arsenic, is characterized by gradual loss of vision, especially centrally At first the disc appears normal but if the trouble progresses or the cause is not removed, optic atrophy will result.

Optic Atrophy. Simple or Primary Optic Atrophy The nerve head is white, with sharply defined edges; diminution in size and some depression may be observed This occurs with pressure on the optic nerve or chiasm, retrobulbar neuritis (often a manifestation of multiple sclerosis), severe toxic amblyopia, neurosyphilis, especially tabes dorsalis, and trauma to the optic nerve.

Postneuritic or Secondary Optic Atrophy This is the end result of papilledema or intraocular optic neuritis The disc is white, has a slightly yellowish tinge (due to gliosis), and somewhat irregular and hazy margins, there is shrinking of the arterioles and, to a large extent, of the veins. In the late stage one may be unable to distinguish between primary and secondary atrophy

Arteriosclerotic Retinopathy. The vascular changes occuring in arteriosclerosis have already been described If the process progresses to the point of inadequate blood supply, changes in the retina occur areas of localized ischemia appearing as white, usually round and clearly defined plaques, hemorrhage, and, if some

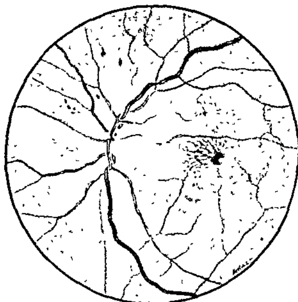


FIG 4 16 Hypertensive and nephritic retinopathy Narrowing and focal constrictions of arterioles, partial "macular star", cotton wool patches, and small hemorrhages From May's *Manual of Diseases of the Eye*, 21st edition The Williams & Wilkins Co, Baltimore 2, Md Reproduced with permission of Dr Charles A Perera

of these are absorbed, areas of retinal atrophy indicated by ill-defined veil-like opacifications spotted with granular pigment These are most often seen around the nerve head and macula Occasionally, closure of a central artery or a large branch will occur, causing marked diminution in the size of the arterioles and pallor of the affected area, except for the macula, which remains red. When the smaller arterioles also become involved in the sclerotic process, the picture becomes similar to that seen in advanced hypertensive retinopathy.

Hypertensive Retinopathy. The wide variety of forms that hypertensive disease can take and the fact that it may exist with or without general arteriosclerosis make a complete account of the various types of retinal change impracticable here As a rule, the earlier stages are marked by signs of diffuse, more *or less uniform constriction of the arterioles and perhaps some arteriolovenous* compression In time, focal constriction of arterioles may appear and, later, focal or generalized arteriolosclerosis In the more advanced stages one finds indications of interference with retinal circulation areas of ischemia, hemorrhage, edema and exudate Finally, choked disc develops (*hypertensive neuroretinopathy*) *The papilledema may be as pronounced* as that seen in elevated intracranial pressure from other causes In the latter, however, the vascular changes are not as striking and hemorrhages and exudates predominate about the disc

The final stages of arteriosclerotic and hypertensive retinopathy are virtually indistinguishable.

Diabetic Retinopathy. Occurring most often in diabetics beyond middle life

and usually associated with arteriosclerosis, but sometimes seen in younger persons, the picture is one of small, round, white or yellowish-white deposits in the retina, small, round hemorrhagic areas, and, in advanced cases, large cotton-wool deposits, especially in the macular region. The appearance often so closely resembles that of arteriosclerotic retinopathy that other data are necessary for differential diagnosis. Eventually proliferation of the gliovascular membrane may cause separation and consequent blindness.

Changes due to Blood Disorders. In the anemias, especially pernicious anemia, pallor of the fundus, hemorrhages of the retina and white deposits similar to those seen in arteriosclerotic retinopathy are not uncommon. In leukemia small hemorrhagic areas, often with white centers, and sometimes edema of the discs is observed. In polycythemia vera or polycythemia secondary to congestive cardiovascular disorders, the retina is dark red, the veins are engorged and the disc borders may be indistinct. Petechial hemorrhages are of importance in the diagnosis of blood stream infection, especially subacute bacterial endocarditis.

THE EARS

The Auricles

Malformation. A small misshapen external ear often with atresia of the meatus is occasionally encountered. It is a congenital defect, usually unilateral. Edema, congestion and perichondrial hemorrhage secondary to repeated trauma result in a swollen, inflamed, thickened auricle (*cauliflower ear*), this is most often seen in pugilists and wrestlers. Ensuing secondary infection may, by destruction of the supporting cartilage, end in deformity, thickening and loss of rigidity.

Displacement. In many normal persons the two auricles stand out away from the sides of the head. This variant is congenital and of no significance except that when pronouced it may be a source of emotional maladjustment. Acquired displacement which is usually accompanied by pain and tenderness is most likely due to postauricular edema. It is an indication of an inflammatory process of the auricle, canal or mastoid. Edema is evident on inspection of the postauricular area, it is not always sufficient to displace the auricle. It is not to be expected with uncomplicated middle ear disease.

Tenderness. Tenderness over the tragus indicates inflammation of the canal, behind the ear it is usually a sign of mastoid disease. In either case it must be distinguished from tenderness due to pre- or postauricular lymphnoditis secondary to infection of the auricle, scalp or other contiguous structure. Uncomplicated middle ear disease rarely produces external tenderness.

Local Lesions. Sebaceous cysts, common in the lobule, appear as discrete, fairly firm, smooth, rounded, movable subcutaneous swellings.

Tophi are yellow to white, 1–3 mm. concretions on the helix, usually its outer margin, which at first glance appear to be on the surface but are actually covered by a thin layer of tissue. They occur only in gout. A tophus must not



FIG. 4 17 Gouty tophi on helix of ear

be confused with the Darwinian tubercle or other irregularity of the auricular cartilage In a doubtful case the identity of a tophus must be established by *microscopic examination of its contents*

KERATOSIS AND EPIDERMOID CARCINOMA often occur on the helix.

THE AUDITORY CANALS

The canal and drum membrane can be examined with an ear speculum and light reflected from a head mirror or with an electric otoscope The latter has *the advantage of magnifying lenses A good view is obtainable only if one pulls* the auricle upward and backward to compensate for the oblique direction of the canal Contact with the bony meatus, which is exceedingly sensitive, must be avoided by inserting the instrument only a few millimeters If the tympanic membrane is not clearly visible, one of the following variants is likely

Cerumen or Desquamated Epithelium.

Foreign Body.

Inflammation of Canal Wall. Most commonly seen are single or multiple furuncles, *cellulitis, eczema and mycotic infection The last is characterized by* profuse discharge and scales of desquamated epithelium

Serous or Purulent Discharge. Discharge indicates otitis media or infection of the external meatus When tenderness is present, *cellulitis or furunculosis* of the canal is likely But tenderness does not exclude middle ear disease, since

the canal may become secondarily infected by material draining from within A foul purulent discharge suggests inadequate care of a chronically discharging middle ear, bone necrosis, fungus infection of the canal, or cholesteatoma.

Blood. The usual causes are local trauma to the external canal and rupture of the drum from trauma or acute hemorrhagic inflammation of the middle ear. Bleeding also occurs in basal fracture of the skull and occasionally is a sign of malignant disease of the external or middle ear.

Benign Polyp. The growth originates in a chronically infected middle ear and extends through a perforation to partially or totally fill the canal

Bony Abnormality. Variations in contour, direction or caliber of the canal are not uncommon congenital aberrations Exostosis of bone, single or multiple, may occur in later life.

The Tympanic Membrane

Although showing considerable variation in different individuals, in general, the drum appears as a shiny, translucent, pearly-gray membrane diagonally placed at the inner end of the canal with its superior posterior portion closer to the examiner's eye than the anterior inferior portion As viewed from the meatus, the drum appears slightly concave, its deepest point corresponding to the tip of the handle of the malleus The examiner is oriented by certain well-defined landmarks: (1) The short process of the malleus, a sharply defined, white, rounded protuberance in the upper anterior quadrant (2) The handle of the malleus, extending backward and downward from this point almost to the center of the drum and appearing as a yellowish-white stripe with a somewhat widened and spatula-shaped tip (3) The light reflex, a bright, shiny light fanning out forward and downward from the tip of the handle almost to the outer edge of the drum on its anterior inferior quadrant.

The drum should be examined for color change, bulging, retraction, variation of the light cone and perforation.

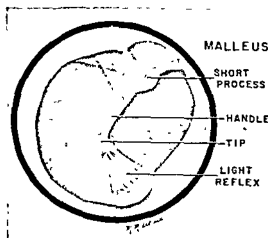


Fig 4 18 Normal tympanitic membrane, right ear

Secretory Otitis Media. Temporary closure of the Eustachian orifice, probably secondary to low-grade infection of the nasopharynx or middle ear, causes a sensation of fulness in the ear and impaired hearing This may be relieved temporarily by shifting the head to another position. The drum is usually pearly-gray and lusterless but sometimes, in the early stages, red and retracted, the normal landmarks are visible With a moderate amount of secretion, a fluid line may be seen across the drum; it will shift with changes in the position of the head If fluid fills the middle ear, the line will disappear and hearing will not be altered by change of position Here the diagnosis depends chiefly on the history and perhaps flattening or slight bulging of the membrane.

Aero-Otitis Media. Presenting a similar picture, this is common in flying personnel, usually associated with rapid changes in altitude, occurring especially in the presence of nasopharyngeal infection, and characterized by rapid accumulation of serous or serosanguineous fluid It can also occur in divers and caisson workers if they are subjected to rapid changes of pressure.

Chronic Catarrhal Otitis Media. As a result of repeated nasopharyngeal and consequent tubotympanic infection or secretory otitis, impairment of hearing and tinnitus gradually develop The drum is dull and lusterless, diffusely or irregularly retracted, the bony landmarks prominent, and the cone of light displaced, distorted or absent

Acute Suppurative Otitis Media. The patient complains of deafness, pain and perhaps tinnitus Redness of the drum appears first, followed by loss of landmarks and later bulging Early rupture is attended by discharge of serous or serosanguineous fluid, later becoming purulent; if rupture does not occur until two or three days after onset, the discharge is purulent from the start A very profuse discharge occurring in a fulminating otitis media and lasting more than a week is strongly suggestive of mastoid infection, even in the absence of post-auricular tenderness, swelling and roentgenologic changes

Chronic Suppurative Otitis Media. Usually following one or more attacks of

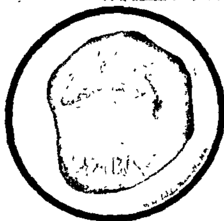


Fig 4 19 Suppurative otitis media, right ear Bulging of drum and absence of normal landmarks

acute suppurative otitis media, and especially common after scarlet fever and measles, the chronic process is characterized by intermittent or persistent purulent discharge. When the material is wiped away, a dark, rounded area of perforation of the drum is disclosed. A rounded, shiny, red mass lying against the drum and movable on probing may be observed, this is a polyp of the middle ear extending through the perforated drum If, as sometimes happens, the drum is almost totally destroyed, one may see the medial wall of the middle ear which, if inflamed, appears red and injected, otherwise of a color not unlike the drum. The ossicles are often destroyed Hearing is impaired but not always entirely absent, since the sound waves may be transmitted directly through the round window.

Hearing

Audiometer tests are the only exact means of determining auditory acuity. For routine purposes, however, ability to recognize words from varying distances is satisfactory. The patient, side towards the examiner, stands 20 feet away with a finger pressed on the tragus of the further ear to close-off its canal and with eyes shut to prevent lip reading. If the nearer ear is normal, he will recognize whispered words at 20 feet. If not, the test is repeated at shorter distances until the point is reached where he first distinguishes whispered words, or in the case of marked impairment, spoken words. His position is then reversed and the other ear similarly tested Hearing for each is expressed in fractions of 20. normal is 20/20, if whispered words are made out only at 10 feet, hearing in that ear is expressed at 10/20, if at 5 feet, 5/20 and so on. If whisper is not heard at a distance of 1 foot, soft and loud spoken words are tried and the results recorded

The tuning fork is used to determine whether diminished hearing is due to a blocked external auditory canal or diseased middle ear, or to a disorder of the cochlea or its tracts Ability to hear a vibrating tuning fork when it is held close to the ear but not touching the head is compared with ability to hear it when its base is pressed against the mastoid process When hearing is normal, the sound is best heard with the fork held beside the ear (*air conduction*) If blocked canal or middle ear disease is hampering transmission of sound waves, the fork will be better heard when its tip is held against the mastoid (*bone conduction*). In nerve or labyrinthine disease, the sound will be poorly heard from both locations In *Weber's test*, the fork is held against the midline of the forehead or occiput, a normal person will hear equally well in each ear. In conduction deafness of one ear, the sound is best heard on the affected side; in perception deafness, on the unaffected side.

Conduction Deafness. The common causes are:

1 Block of the external canal, most commonly impacted cerumen, inflammatory swelling, tumor or, especially in children and psychotics, foreign body.

2 Acute or chronic disease of the middle ear or Eustachean tube.

3 Otosclerosis, a hereditary disturbance most common in women, and characterized by fixation of the stapes due to new bone formation

Perception Deafness. Here the trouble is in the labyrinth or nerve tracts. Common causes are:

1. *Any focal or general toxemia*
2. Certain acute infectious diseases, particularly mumps and cerebrospinal meningitis
3. Syphilis, congenital or acquired
4. Otosclerosis in its late stages. Here there is probably impairment of the nerve pathways in addition to the bony change noted above
5. Leukemia
6. Tumor of the cerebellopontine angle or eighth nerve.
7. Congenital defect
8. Fracture of the skull involving the eighth nerve or structures of the inner ear
9. Prolonged exposure to constant noise (*boilermaker deafness*) or to sudden concussion from a loud blast (*blast deafness*)
10. Large or continued doses of certain drugs, such as quinine and its derivatives, salicylates and streptomycin

THE NOSE

General Appearance

Redness, especially when associated with dilated capillaries or venules, is popularly and often correctly attributed to alcoholism, but the same appearance is frequently due to exposure to the elements and can occur without known cause. Generalized or patchy redness is also encountered in a number of dermatologic disturbances which have a predilection for the face, especially acne rosacea, lupus erythematosus, lupus vulgaris and erysipelas. Increased sebaceous secretion in the nasolabial folds, often with plugs of inspissated material, is seen in persons with oily skin; it is sometimes a sign of riboflavin deficiency. Infection of a hair follicle or fissure on an ala nasi or within the vestibule may produce redness, swelling and tenderness of the whole nose, indicating severe cellulitis; if not recognized or properly treated, this may end in some serious complication such as cavernous sinus thrombosis or meningitis.

Enlargement. Hypertrophy of the skin and sebaceous glands, which may be sufficient to give the nose a bulbous appearance, is seen in the late stages of acne rosacea (*rhinophyma*). In acromegaly the entire structure is enlarged; in myxedema, subcutaneous infiltration alters the normal outlines.

Depression of Bridge. This suggests congenital syphilis of the nasal bones. It is also seen as a sequela of trauma, or septal abscess following trauma, but may be found in the absence of disease or injury.

Nodularity and Ulceration. Often accompanied by scarring and atrophy, these are seen in lupus vulgaris, late syphilis, leprosy, and other granulomatous diseases. An indolent area of thickening or ulceration, especially if near an eye, suggests epidermoid carcinoma or tuberculosis.

Dilatation of Nostrils. In disturbances producing exaggerated breathing, such

as pneumonia and other acute infections, serious heart disease and severe emotional upset, the alae nasi move outward with each inspiration

Nasal Discharge. This almost always appears externally but may be discovered only on inspection of the nares A *watery, mucoid* discharge occurs in acute coryza and vasomotor rhinitis, producing redness and irritation of skin around the nostrils. Nasal diphtheria is suggested by *thin, watery* secretion, especially if *blood tinged* and associated with excoriations and crusting about the orifices. In acute or chronic sinus infection one observes *thick, yellow, mucopurulent* material, unilaterally or bilaterally Foreign body is suggested by a similar discharge, especially if foul and unilateral. Atrophic rhinitis is indicated by the appearance of *green* or *yellow-brown* malodorous crusts and mucoid casts

Nosebleed. Usually evident externally, blood may be found only on examination of the nares or pharynx, especially in a feeble, bedridden patient; recent bleeding is indicated by dried blood The most common cause of nosebleed is direct trauma. Spontaneous bleeding is most often due to minor excoriation of superficially placed blood vessels on the cartilaginous septum, usually in the anterio inferior area It is also common in acute coryza or other nasal infection, hypertension, diseases causing hemorrhagic diathesis, such as leukemia, nephritis, purpura, hepatitis, chemical poisoning and deficiency states, in ulcerative or malignant disease of an intranasal structure and following sudden changes in atmospheric pressure, as in deep-sea diving and high-altitude flying. It is not uncommon during the earlier stages of typhoid fever, and is regarded as an important sign of activity in rheumatic infection

The Nasal Cavity

A satisfactory view of the anterior nares and anterior part of the septum can be obtained with the aid of a flashlight, but for their midportions a nasal speculum and reflected light are required It is preferable to have the patient sitting up with his head erect, not tipped forward or backward With the flashlight one can observe medially the smooth, pinkish mucous membrane of the anterior third of the septum and laterally the anterior third of the lower turbinate, which appears as a pink, rounded, smooth protuberance With the speculum can be seen about two-thirds of the septum, all of the lower turbinate, the region above and behind it, and the anterior half of the middle turbinate If the lower turbinate is swollen, as from acute coryza, shrinkage with some vasoconstricting agent will be required in order to obtain a satisfactory view The posterior nares can be seen only with the electric pharyngoscope, the use of which requires special training

Anosmia. This is common in acute coryza, vasomotor rhinitis, atrophic rhinitis, and especially when the nose is obstructed by polyps in chronic sinus infection It may occur without known cause and is also encountered in certain neurologic disturbances (see Chap 36)

Obstruction Each naris is tested separately The patient attempts to inhale through one side while holding his finger over the other nostril. Closing the latter by pressure on its lateral wall must be avoided; this procedure may force

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Dilatation of Nostrils. In disturbances producing exaggerated breathing, such

Transillumination

This method of examination is useful when disease of an anterior accessory sinus is suspected. A dark room is required. A small electric flashlight is held in the patient's mouth with his lips closed around it and the light directed toward the medial wall of the antrum. If the antrum is normal, a glow will be *transmitted, appearing as a crescent between the floor of the orbit and the eye;* the pupil may appear red and the patient will perceive light in the eye. If the antrum is badly diseased, the light fails to penetrate Another method is to place the lamp between the cheek and the posterior portion of the alveolar process behind the canine fossa. If the antrum is normal, light will appear through the floor of the orbit. Transillumination of the frontal sinus is not as satisfactory. The lamp is placed against the roof of the orbit above the inner canthus; through a normal sinus, light will appear in the forehead

The frequency of developmental abnormalities of bone or sinus limits the value of transillumination, it should not be relied upon without other evidence of sinus disease. In a doubtful case or when trouble in a posterior sinus is suspected, x-rays must be taken

THE BREATH

Foul Breath. This is most often due to some local cause such as:

1. *Unhygienic mouth, especially when there is associated parodontal disease*
2. Stomatitis from any cause
3. Follicular tonsillitis with deposits in the crypts
4. Intranasal disease, especially atrophic rhinitis or foreign body
5. Abscess or gangrene of the lung or severe bronchiectasis.

Acetone Breath. One notes a faintly sweetish odor which has been variously compared to that of chloroform, new-mown hay and rotting apples It occurs in acidotic states as in diabetes, severe malnutrition and dehydration.

Uremic Breath. Often observed in patients with uremia, the breath has an ammoniacal odor or one resembling that of urine In incontinent patients care must be taken not to mistake the odor of urine-soaked clothes or sheets for uremic breath.

Mousey Breath. A curious musty odor, best described as "mousey", can sometimes be detected in severe liver failure

Alcoholic Breath. Although one should be wary *of the statements of its* possessor, *an alcoholic breath does not always indicate intoxication.* In a stuporous or unconscious person, it does not necessarily indicate that alcohol is the cause of the trouble; the patient may have had a drink shortly beforehand or, following onset of stupor, may have been given one as an emergency measure by a misguided bystander

Following the use of paraldehyde and certain other drugs and *in cases of* illuminating gas inhalation, the odor of the substance involved may be detectable on the patient's breath

the lower septum toward the opposite side, thus causing artificial obstruction of the naris which is being tested. Common intranasal causes of *bilateral* obstruction are acute rhinitis from any cause, hypertrophy of the lower turbinates from chronic intranasal infection or irritation, and S-shaped septal deviation. Occasionally free flow of air is hampered by narrowing of the vestibules due to impaired development of the lower lateral nasal cartilages. *Unilateral obstruction is most likely due to deviated septum, polyp, foreign body, or malignant disease.*

Acute Rhinitis. Also known as *acute coryza* or *common cold*, this is characterized by hyperemic mucous membrane, swollen turbinates, and a thin watery discharge which later becomes thick and mucoid. A similar picture is encountered at the onset of certain exanthematous diseases, especially measles.

Vasomotor Rhinitis. In contrast to acute coryza, the mucous membranes and turbinates are swollen but pale. A profuse, watery discharge is present.

Chronic Hypertrophic Rhinitis. Resulting from repeated attacks of acute rhinitis, this shows thickening and injection of the mucous membrane, enlarged congested turbinates, and narrowed airways.

Chronic Atrophic Rhinitis. The turbinates are atrophied, the nasal cavities wide, and the mucous membrane dry and sheenless. The septum, floor of the nose, and turbinates are partially covered with dry, greenish or yellowish-brown, easily removable crusts which have a characteristic foul odor readily discernible on the breath.

Septal Perforation. *This may be produced by trauma from constant picking,* a chronic destructive infection such as syphilis, tuberculosis or lupus, and occasionally by operation on the septum. If the lesion involves the bony septum as well as the cartilaginous portion, it is almost certainly due to syphilis; if the bony portion is not involved, syphilis is probably not the cause.

Septal Deviation. Although often a developmental anomaly, gross deviation is frequently a result of direct trauma. When pronounced, it interferes with ventilation on one or both sides and may predispose to sinus or middle ear infection.

Septal Hematoma. Not uncommon after trauma, especially in children, this is characterized by red, bilateral swelling of the septum sufficient to cause pronounced obstruction to the airways. *Failure to recognize this picture and to treat it properly may result in suppuration, with destruction of the cartilaginous portion and consequent depression of the nasal bridge.*

Polyp. Polyp appears as an edematous, saccular mass of tissue covered with pallid, boggy mucous membrane. It arises in a chronically infected sinus and is often multiple. The possibility of malignant disease must be borne in mind; biopsy may be necessary for diagnosis.

Foreign Body. Entrance is usually through the anterior nares but occasionally through the posterior as a result of vomiting or disturbance of the swallowing reflex. It should be suspected in any case of unilateral, purulent discharge, especially in children.

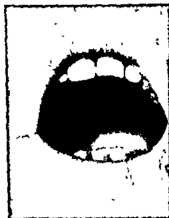


FIG 4 20 Angular cheilosis due to prolonged use of ill fitting dentures

as the various forms of stomatitis, may spread to the lips Certain cutaneous diseases, including lupus vulgaris, lupus erythematosus, eczema, lichen planus, pemphigus and erythema multiforme, may involve the vermilion parts In children, serous discharge and crusting suggest impetigo

Fissures. These are common in diffuse inflammation from any cause. Inflammation at the commissures usually with maceration and transverse fissuring (*angular cheilosis*) is most likely due to excessive salivation from some irritating process in the mouth such as an ill-fitting denture, or stomatitis The process may extend downward and outward over a few millimeters of adjacent skin. It is also an important sign of deficiency states, especially B-complex deficiency, pernicious anemia and primary hypochromic anemia When the fissuring is especially persistent and extends over adjacent skin, syphilis is suggested

White linear scars radiating from the commissures are often healed syphilitic lesions, most likely of congenital origin.

Mucous Patches. (See Tongue)

Herpes Simplex. The lesions, popularly known as *cold sores* or *fever blisters*, appear most commonly on the lips but are found also on the cheek, nose, or chin Sometimes they form in the mouth, less commonly on other parts of the head or body The eruption is usually a cluster of pinhead-sized or slightly larger vesicles which dry within a few days, leaving a yellowish crust which gradually disappears Distribution is usually unilateral Presumably these lesions, when occurring on the face, are caused by inflammation of the Gasserian ganglion Herpes is particularly apt to occur in connection with acute coryza, pneumonia, meningitis and malaria, and is also common in association with acute sunburn, especially when the latter was acquired at the beach or on the water.

Keratosis. On the lip keratosis appears as a dry, hard, flat, whitish, scaling lesion It is usually circumscribed but non-indurated and bleeds easily if disturbed It is potentially malignant

Leukoplakia. This has the same character and significance as leukoplakia

EXAMINATION OF THE MOUTH

Since some changes are better seen in daylight than with direct or artificial light, the former should be used whenever possible. Dentures should be removed One must inspect the lips, the vestibule, teeth, tongue, gingivae, roof, floor and lateral aspects of the mouth, the uvula, pillars and pharynx. The vestibule can be seen by everting the lips, each lateral wall by pushing the cheek outward with a tongue depressor, and the floor of the mouth by having the patient elevate the tip of the tongue. One looks for deformities, changes of color, edema, cracks, membranous deposits, ulcerations, new growths and other variants. Any lesion which creates destruction of tissue is likely to be covered by a whitish patch or membrane. Some disorders have a predilection for certain sites; others show generalized distribution

Alteration of texture characteristic of leukoplakia, the induration of cancer, tenderness, cystic swelling and many masses can be discovered only by digital examination With care even the nasopharynx also can be palpated and lesions that are not visible can be discovered To avoid possible infection of the palpating finger, a glove should be worn In children or sensitive, irrational or unconscious patients, biting must be prevented by placing a mouth-gag between the teeth

THE LIPS

Color. Pallor and cyanosis are often discernible on the lips before they become evident on the skin Both are frequently overlooked because of cosmetics

Parted Lips. Dyspneic patients, those with hypertrophied adenoids or nasal obstruction, and some healthy persons facilitate intake of air by breathing through the mouth

Enlargement. In myxedema, cretinism and acromegaly, the lips appear enlarged and thickened

Cheilosis *Discrete* superficial denudation of the epithelium of the lip occurs most frequently from minor trauma, as by a cigarette or pipe-stem Ulceration is most likely the result of irritation by a jagged tooth or ill-fitting denture, but may be due to one of the more serious disturbances listed in the subsequent paragraphs Acute cellulitis, indicated by swelling, redness and tenderness, furuncle or carbuncle, most common on the upper lip, has serious potentialities, if not promptly and adequately treated, infection may involve the cheeks and, by spreading through the regional veins, cause septic thrombosis of the cavernous sinus

Diffuse inflammation, characterized by dryness, swelling, and perhaps serous discharge, crusting and fissuring, is commonly encountered following sunburn or exposure and in any chronic or febrile illness. Swelling, redness and often superficial ulceration are important signs of nutritional deficiency, the process being, generally speaking, similar to that seen on the tongue or buccal mucosa In older persons, especially women—perhaps as a result of deficiency—the vermilion borders may become thin, dry and wrinkled Lesions of the mouth, such





Fig 4 23 Fig 4 24

Fig 4 23 Partial harelip (Courtesy Dr V H Kazanjian)
Fig 4 24 Complete harelip and cleft palate (Courtesy Dr V H Kazanjian)

the structures on the two sides to join properly during fetal life It may be limited to a small cleft involving only the vermilion part, extend to the nostril, or even be contiguous with an anterior-posterior cleft through the hard palate (*cleft palate*) The defect may occur on one or both sides In the latter instance there is, between the two fissures, a small island of tissue continuous with the nasal septum

THE TONGUE

The tongue must be observed not only for the indications of local disease but also for those of some systemic disturbance

Tremulousness Nervousness, thyrotoxicosis, alcoholism, dementia paralytica and drug addiction are suggested. A hesitating tremulousness is associated with old age, typhoidal states, encephalitis, other types of prolonged infection and wasting diseases

Lateral Deviation When the tongue is actively protruded, slight deviation from midline is seen in many persons, especially those who lack several teeth on one side In hemiplegia, deviation is greater, the tongue is directed away from the lesion and toward the paralyzed side In disorders of the hypoglossal nerve or its nucleus, as in bulbar paralysis or tabes dorsalis, it deviates toward the side of the lesion Contraction of a scar from such a cause as injury or operation will pull the tongue toward the side A deep-seated tumor not readily seen or felt may be indicated by deviation of the tongue to one side due to involvement of the contralateral peripheral nerve

Coating. Due mostly to lack of saliva or to evaporation, as in mouth breath-



Fig 4 21 Epidermoid carcinoma of lip



Fig 4 22 Chancre of lip

elsewhere (see Tongue). On the lips it appears as diffusely distributed, whitish patches of thickened epithelium

Epidermoid Carcinoma. Epidermoid carcinoma and chancre should be suspected whenever a lesion on the lip lasts more than 10 days to 2 weeks The former occurs almost always on the lower lip, and in males past middle life In the early stages there is marked disk-like induration. The surface may be warty, crusted, or centrally ulcerated The lesion as a rule is almost insensitive and is sharply defined from the surrounding healthy tissue The margin may present an elevated, pearly, translucent zone, as does epidermoid carcinoma elsewhere. In the later stages, there is often a demonstrable loss of substance with leakage of saliva and possibly a considerable amount of inflammatory reaction in adjacent tissues Regional lymphnode enlargement appears slowly Involved nodes are hard and shotty

Chancre. Less well-defined at its edges than epidermoid carcinoma, chancre assumes an inflammatory character early in its progress, ulcerates promptly, and gives rise to a serous or serosanguineous exudate The sore lasts a few weeks, and if observed from week to week, will be seen to alter its character. The regional lymphnodes are involved almost immediately and tend to be larger and softer than in carcinoma *Dark-field examination of the exudate and serologic tests should always be made in the case of any suspicious lesion of the lip*, especially when a so-called "cold sore" has lasted for more than a few days or a surgical procedure is *contemplated for what is believed to be carcinoma*

Angioneurotic Edema. The whole lip suddenly swells, often to huge size It feels stiff but is not painful Diagnosis depends on the exclusion of such other causes as trauma, infection and insect bite A history of previous similar swelling *here or elsewhere is confirmatory evidence.* In contrast to that due to other causes, the edema usually disappears within a few hours A similar picture may occur as a part of generalized urticaria.

Harelip. This congenital deformity of the upper lip is caused by failure of

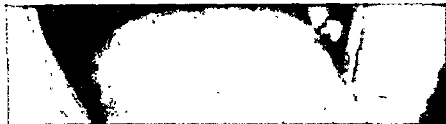


FIG 4 26 Enlarged papillae of tongue in scarlet fever, 2 days after onset of eruption. (Courtesy Dr. Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

red, standing out against the white surface. Later, the white coat disappears, leaving a bright red surface as a background for the enlarged papillae, thus giving the strawberry appearance. Although valuable in the diagnosis of scarlet fever, this picture can occur in glossitis from other causes.

Large Tongue. *General* enlargement is seen in Mongolian idiocy, cretinism, adult myxedema, acromegaly, and, rarely, in amyloidosis Diffuse swelling occurs in glossitis, stomatitis, regional inflammatory disturbances such as Ludwig's angina, or cellulitis of the upper neck, and, because of interference with lymphatic or venous drainage, in mediastinal tumor or malignant disease of cervical lymphnodes An acutely swollen tongue without evidence of infection suggests angioneurotic edema. *Asymmetrical* enlargement is seen with congenital benign tumor such as lymphangioma or hemangioma

Indented Edges. The tongue border may be indented by a malposed or jagged tooth, especially in a neglected mouth Edema is suggested when the indentation shows a pattern made by a series of adjacent teeth.

Furrows. Frequently found on the dorsum, furrows are fine linear depressions,



FIG 4 27 Congenitally fissured tongue (Daughter and granddaughter of this patient have tongues of similar appearance) (Courtesy Dr David Weisberger)

ing, a coated tongue has less clinical significance than is popularly accorded it, especially by apprehensive mothers Many healthy persons, especially mouth-breathers, smokers and those who keep late hours, have coated tongues on arising. Appearance of a coat on a usually clean tongue may be associated with a digestive disturbance, constipation or fever A darkly coated tongue is said to be caused by a fungus infection of the papillae or to piling of epithelial elements associated with impaired motility or deficient salivation It may also occur during prolonged use of an antibiotic.

Dryness. The tongue is an invaluable index of the state of body hydration. In prolonged fevers, toxic states, serious vomiting or diarrhea, exhausting and wasting disease, and dehydration from other causes, it is frequently dry and shriveled, indicating need for increased fluid administration. It should not be forgotten however, that dyspnea or mouth breathing can also produce dryness but here the organ does not appear shrunken as in general dehydration

Smoothness. Usually due to atrophy of the papillae, smoothness is a characteristic finding in pernicious anemia, idiopathic hypochromic anemia, chronic gastro-intestinal disorders, especially those with achlorhydria, gastric carcinoma, sprue, pellagra, and other deficiency states. Smooth tongue is usually pink, but may be almost salmon-colored in severe anemia, red in pellagra or when inflammation is present. It is likely to be sore. In vitamin A deficiency, the smoothness is patchy and interspersed with islands of hypertrophied papillae The tongue of *Plummer-Vinson* syndrome is smooth, small and wrinkled The buccal mucosa shows similar changes When any of the above disorders is advanced, generalized atrophy—indicated by small size and wrinkling—is observed

Strawberry Tongue. In early scarlet fever the tongue has a white coat, except at the tip and along the edges, where it is red, the papillae are enlarged and



FIG 4 25. Smoothness of tongue and atrophy of its papillae due to nutritional deficiency Angular cheilosis also evident. (Courtesy Dr David Weisberger)



FIG. 4.28 Leukoplakia on side of tongue in a constant pipe smoker (Courtesy Dr David Weisberger)

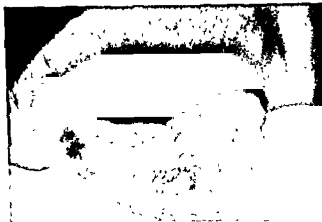


FIG 4 29 Chancre on tip of tongue (Courtesy Dr David Weisberger)

Whitish streaked lesions and sometimes large thick plaques resembling leukoplakia are seen as part of the lichenoid dermatitis of quinacrine sensitivity.

Syphilis. The tongue may be involved at any stage of the disease

CHANCRE is usually a smooth eroded plaque with considerable induration apparent on palpation It resembles chancre of the lip.

MUCOUS PATCHES, the typical mucous membrane lesions of secondary syphilis, appear on the tongue, buccal mucosa and lips They are slightly raised, round or oval, mildly eroded areas covered by pearly-gray membrane They vary in diameter from 5–10 mm. On the side of the tongue, they may be deeper and show a tendency to ulcerate, on the dorsum erosion is rare Several lesions may become confluent, giving a smooth, flat plaque In contrast to simple canker sores, they are less painful, more raised and indurated, have a "buttony" feeling, and are usually accompanied by cervical lymphnoditis.

often congenital, sometimes familial; they do not necessarily indicate disease. They must be distinguished from the wrinkles due to atrophy or dehydration Inflammatory furrows may occur in tongues which are the seat of chronic superficial inflammation Deep fissures suggest syphilis Chronic irritation from jagged teeth or a dental appliance may cause one or more furrows with eventual scarring Scars also result from involuntary biting during falls or convulsive seizures.

Geographic Tongue. Circinate sinuous areas of heaped-up epithelium surround areas of atrophy, giving a red tongue on which the white raised areas stand out in relief The outlines change frequently as the zones of piled-up epithelium and atrophy shift position A superimposed mild inflammatory process may give rise to a burning sensation The picture may be seen in dietary deficiency.

Pigmentation. In some normal persons, especially of the darker races, the tongue may show one or more small sharply defined bluish-black places of pigmentation The cause is not known Similar spots also occur in Addison's disease.

Glossitis. The various diffuse inflammatory disturbances which affect the buccal mucosa usually involve the tongue and often the lips (see Buccal Cavity) Depending on the cause, one may find redness and swelling of the papillae, generalized swelling, or local areas of inflammation and ulceration The tongue may become swollen, red and painful as an allergic response to a drug, less often to some other allergen

Occasionally on the lingual margins just forward of the anterior pillars, areas of soreness, redness and slight swelling will appear, these are islands of lymphoid tissue (*lingual tonsils*) inflamed by low-grade infection or irritation from a dental appliance On the posterior third of the tongue, development of small, red, painful and tender nodules indicates inflammation of the circumvallate papillae In older persons, small varices are a not infrequent cause of discomfort beneath the lateral lingual margins

Herpes. The lesions of herpes may appear on the tongue or buccal or palatal mucosa as clusters of vesicles, usually unilateral, which soon break down to form shallow areas of ulceration

Abscess. Usually following trauma, abscess appears as a localized, tender swelling with pronounced deformity resulting from edema and infiltration of the deeper structures

Leukoplakia. The sites of predilection are the tongue, lips and buccal mucosa, but any part of the oral mucous membrane may be involved Leukoplakia appears as whitish, opaque patches of proliferated epithelium, smooth in the early stages and later becoming thickened, dry, fissured and lichenified It may be limited to a point of irritation or diffusely distributed over a wide area Margins may or may not be well-defined The course is chronic and painless, unless, probably as a result of local irritation, a patch partially sloughs off, exposing a raw surface. Epidermoid carcinoma may be the end-result The likelihood of the disease is greater in heavy smokers



FIG 4 32 Carcinoma of tongue, left side Raised indurated lesion with central necrosis Anterior to it is an area of leukoplakia (Courtesy Dr David Weisberger)

areas of leukoplakia and intervening smooth, atrophic, scarred areas. Normal papillary structure is almost entirely absent. Deep fissures may occur in the leukoplakic areas. *The incidence of superimposed carcinoma is extremely high; every patient with syphilitic glossitis should be carefully and frequently checked for this complication.*

Tuberculosis. Secondary to pulmonary tuberculosis, the lesion begins as a cluster of small nodules on the dorsum, margin or tip of the tongue. They develop into an irregular, indolent ulcer covered with a dirty-gray membrane, the induration typical of cancer is lacking Intense pain is characteristic. More than one ulcer may develop Involvement of regional lymphnodes is less likely than in carcinoma or syphilis It is often impossible to differentiate between syphilis, cancer and tuberculosis without the aid of serologic tests and biopsy.

Carcinoma. The appearance is very similar to carcinoma of the lip. Common sites are the margins and tip In the late stages motility is impaired by invasion of intrinsic muscles

THE GINGIVAE

Both the lingual and labial aspects of the gums should be examined Normal gingivae are firm, pink, and at their free margins in close approximation to the necks of the teeth, the interdental papillae extend well toward the biting surfaces. Redness, swelling and tendency to bleed indicate gingivitis Pallor and cyanosis have the same significance as in any mucous membrane.

Sordes. When the mouth is abnormally dry, as in febrile states or diminished salivation from such causes as parotitis or regional radiation therapy, dark-brown, foul material—a collection of epithelium, bacteria and food particles—accumulates rapidly about the necks of the teeth. If neglected, sordes may end in gingivitis

Gingivitis. This is indicated by redness, swelling, tendency to bleed and perhaps local pain or discomfort, all or parts of the gingivae of one or both jaws



FIG 4 30 Diffuse glossitis in late syphilis Papillary atrophy, patchy areas of leukoplakia, scarring, and fissuring (Courtesy Dr David Weisberger)

GUMMA, a nodular, deep or ulcerated lesion, usually on the posterior third of the dorsum, occurs as a manifestation of late syphilis There may be fine contractions of scar tissue and deep fissures or furrows with irregular-shaped areas of normal lingual tissue between them

GLOSSITIS, without definite gummatous formation, is another manifestation of late syphilis The normal structures of the dorsum are replaced by patchy



FIG. 4 31 Tuberculosis of tongue in a patient with active pulmonary tuberculosis (Courtesy Dr David Weisberger)



FIG. 4 34 Necrosis of gingivae with extension of process to adjacent buccal mucosa due to Vincent's infection (Courtesy Dr David Weisberger)

GENERAL NECROSIS is more likely due to severe Vincent's infection, blood dyscrasia, deficiency state or granulocytopenia from any cause. Here also the process is likely to begin at the site of tooth extraction or other trauma, perhaps nothing more than a scratch from a toothbrush or food particle.

Atrophy. LOCAL RECESSION of the gum away from the teeth is most likely due to trauma to a single tooth from improper bite causing destruction of underlying bone

GENERAL RECESSION may result from advancing age, incorrect brushing, or chronic gingivitis from any cause

Hypertrophy. LOCAL HYPERTROPHY, indicated by enlargement of the interdental papilla or the entire gingiva in contact with the tooth, is usually the result of irritation from a jagged tooth or ill-fitting crown, it is common in children wearing orthodontic appliances The growth is irregular in shape, redder than normal gingival tissue, and bleeds easily When pain or some other disturbance causes chewing to be restricted to one side of the mouth, the resultant absence of normal massaging of the gums on the affected side may end in unilateral swelling simulating hypertrophy.

GENERAL HYPERTROPHY may be familial and of no clinical significance It also occurs in some cases of pregnancy (*gingivitis gravidum*), and may progress to actual tumor formation of a benign nature (*pregnancy tumor*) which may or may not dis[illegible]ar after parturition In leukemia the gingivae may have a swollen, spongy, [illegible]sh appearance as in scurvy, show a marked tendency to bleed, and on microscopic examination are found to be infiltrated with leukemic cells. Hypertrophy may follow the therapeutic use of diphenylhydantoinate; sometimes its cause is not discoverable

Chronic gingivitis with resultant engorgement and bleeding may be super imposed on a hypertrophied gum

Vincent's Infection. Only the gingiva around a few teeth may be affected, or

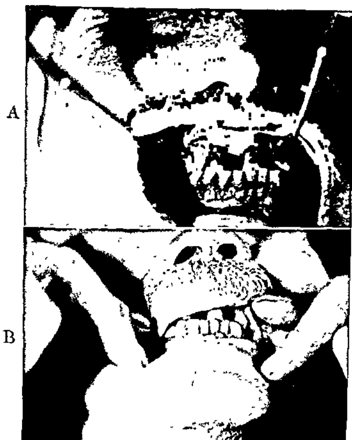


Fig 4 33 Gingival changes in scurvy A Before treatment B After therapy with vitamin C No local treatment

may be affected When the changes are limited to the margins the cause may be entirely local, such as improper chewing or brushing, debris and tartar about the teeth, rough or badly fitting fillings, dentures or appliances, or poor occlusion or bite Deeper involvement may represent progression of the inflammation due to these causes, but may also be seen as part of a general stomatitis, Vincent's or other oral infection It occurs in deficiency states, blood dyscrasias and metallic poisoning In scurvy, swelling and bleeding are the outstanding signs, in avitaminosis B, ulceration predominates In mild cases of infectious mononucleosis marginal gingivitis and inflammation of the interdental papillae occur; in severe cases the process may progress to extensive gingivitis and stomatitis In either event, the appearance of the gingivae resembles that of Vincent's infection

Necrosis. If the gingivitis becomes more severe, ulceration and sloughing occur Sometimes large areas of the gingival and buccal mucosa are involved Bleeding is common Necrotic debris and decomposed blood create a foul odor

Local necrosis may result from regional irritation or tooth extraction or be superimposed on a malignant lesion of the gingival mucosa, often unrecognized in the early stages.

similar organisms may occur as secondary invaders in any necrotic buccal lesion

Acute Vincent's infection is often accompanied by swelling and tenderness, first of the submaxillary and submental lymphnodes, later of the cervical lymphnodes. Severe infection may show fever, malaise, and, in contrast to necrosis due to agranulocytosis, an elevated white count.

Although often secondary to trauma, poor hygiene, dietary deficiency and other causes of simple gingivitis, Vincent's infection also occurs in epidemic form, especially in schools and institutions, being readily transferable by direct contact and eating utensils

Pyorrhea Alveolaris. In long standing gingivitis from any cause, especially poor oral hygiene or chronic Vincent's infection, pocket formation and suppuration between the necks of the teeth and adjacent gingiva may develop Pus can often be seen along the gingival margin or can be expressed from the pocket by light pressure on the gingiva below. Extension of the process into the deeper structures around a tooth results in destruction of the supporting bone and consequent loosening of the tooth. In advanced cases, the gingiva surrounding the neck and root of the tooth as well as the overlying bone are destroyed, exposing the root Roentgenologic examination shows horizontal bone resorption about the tooth; when infection extends downward along the root, the peridental space is widened and the cortical bone surrounding the root destroyed If the pus-pocket becomes walled off and drainage ceases, a gingival abscess forms near the gingival margin, this is one form of so-called *gumboil.* The other form of gumboil occurs as a level nearer the apex of the tooth root as a result of break through or pointing of an alveolar abscess (see Teeth)

Metal Line. Examination of the lingual as well as the labial aspects of the gingivae is of particular importance when searching for any metallic line

PLUMBISM This often shows, as an important diagnostic feature, what appears to the naked eye as a dark blue-to-black scalloped line following the undulations of the gingiva near its junction with the necks of the teeth Examination with a magnifying glass shows this line actually to be made up of a series of closely approximated pinpoints of pigment This *lead line* appears only about the necks of dirty teeth It can easily be confused with the dark line of black tartar which is so often deposited on neglected teeth just below the gingival margins and is visible through the overlying gingival tissue. If a small, wedge-shaped piece of paper is inserted between the gingiva and the tooth, a lead line will appear in sharp relief against it; tartar, which lies on the tooth surface, will be hidden If the diagnosis cannot be established in this way, a tiny piece of gingiva may be cut from the discolored area, crushed between glass slides, and examined microscopically If lead deposit is present, the pigment will be visible

BISMUTH, MERCURY Bismuth, mercury or other heavy metal absorption may cause discoloration similar to that of lead line but the staining is apt to be more diffuse and to occur at some distance from, as well as close to, the free gingival



FIG 4 35 Gingivitis gravidum with pregnancy tumor (Courtesy Dr David Weisberger)



FIG 4 36 Swollen gingivae in a case of monocytic leukemia (Courtesy Dr David Weisberger)

the disease may be widespread, not only in the gums, but elsewhere in the mouth The buccal mucosa, sublingual structures, uvula, tonsils or peritonsillar regions may be either secondarily involved or the seat of the primary infection In the gums, the early picture is that of simple gingivitis, but is apt to start in the interdental papillae and produce more pronounced pain, tenderness and bleeding Later, wider areas become involved and the gingivae are covered with a grayish-white slough easily wiped off and revealing an inflamed, bleeding, ragged base beneath The slough must not be mistaken for sordes, which, when removed, bares a fairly normal mucosa, or for the film of thrush, which is difficult to remove In contrast to gingivitis due to other causes, necrosis and ulceration are usually first observed in the interdental papillae Except in advanced cases, other parts of the gums, although perhaps inflamed, are [illegible] necrotic, this characteristic alone is not sufficient to differentiate Vincent's [illegible] other forms of severe gingivitis Furthermore, the characteristic foul odor is out of proportion to what the physical signs would lead one to expect. Large numbers of fusiform bacilli and spirochetes will be found on stained smear of the exudate Although highly suggestive, this finding is not necessarily confirmatory, since

Fibroma. One observes a soft or hard, circumscribed, slow-growing benign tumor of the gingival or buccal mucosa, covered by normal-appearing mucous membrane. It is sometimes pedunculated

Malignant Tumor. Lymphosarcoma, fibrosarcoma and epidermoid carcinoma can originate in the gingiva or deeper supporting structures. It appears as necrotic ulcerating lesion easily confused with a local area of Vincent's infection. On careful examination, however, the malignant process will show, marginally or beneath the slough, some degree of tissue proliferation.

THE TEETH

The number and condition of the natural and artificial teeth and the total area of functioning chewing surface should be noted. Improper mastication due to inadequate chewing surface, ill-fitting denture or local pain may account for minor digestive complaints

The eruption time of the first or deciduous teeth and the second or permanent teeth is indicated in Figures 4.39 and 4 40. The time and order of appearance of the deciduous teeth is more uniform than that of the permanent teeth.

Delayed Dentition. Tardy eruption or x-ray evidence of slow calcification of crown or roots is one of the cardinal signs of developmental disorders such as cretinism, rickets, or, perhaps, Mongolian idiocy. Failure of one or more teeth to appear may, however, be due to impaction or, less often, absence of the tooth germ.

Malposition. Because of faulty position, some teeth may erupt only partially or not at all Infection of tissues around a partially erupted tooth may cause *trismus* (locking of the jaw) by irritation or collateral edema of the muscles of mastication An unerupted tooth, especially when wedged against an adjacent root (*impaction*), may cause headache or neuralgia Early loss of deciduous

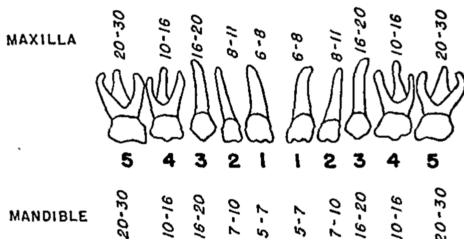


Fig 4 39 Deciduous teeth Numbers immediately below teeth refer to their names 1, central incisor, 2, lateral incisor, 3, cuspid, 4, first molar, 5, second molar Other numbers indicate eruption time in months.



FIG 4 37 Soft fibroma of gingiva with chronic inflammation. (Courtesy Dr Kurt H. Thoma)

margin Bismuth line is often seen in patients undergoing antisyphilitic treatment with this metal.

Benign Giant-cell Tumor. This originates in the peridental membrane, usually near the surface, and appears early as a marginal tumor It is soft, circumscribed, sessile or pedunculated, sometimes lobulated Some observers limit the use of the term *epulis* to this type of growth Others apply it to any benign tumor of a peridental structure A similar growth, known as *brown tumor,* occurs in hyperparathyroidism, it originates deep within the bone and at first can be detected only by x-ray. Later it may extend to the gingiva and, by causing the latter to bulge, be confused with the less significant giant-cell tumor which, as just noted, originates in the peridental membrane nearer the gingival surface Since peridental tumor may be the first sign of hyperparathyroidism, it should in every case be studied with this possible cause in mind.



FIG 4 38 Lymphosarcoma of gingiva (Courtesy Dr David Weisberger)



FIG 4 42 Attrition of teeth presumably due to nocturnal grinding (Courtesy Dr. David Weisberger)

Attrition. Wearing-down of the biting surfaces may merely obliterate the normal surface contours or advance to the point where the biting surface becomes level with the gingival lines Constant chewing on a hard substance, such as a pipestem, is the commonest cause. The necks of the teeth may be worn by improper brushing or chemical action (*erosion*). The latter is thought to be associated with hyperacidity of secretions in the mouth, but this has never been proved.

Hypoplasia of Enamel. Defective enamel, characterized by horizontal striae or pitting, is often traceable to nutritional disorders and febrile diseases of childhood Brown staining and pitting (*mottled enamel*) are commonly encountered, especially in children, in regions where the drinking water has a high fluorine content. In children, use of too much fluorine in therapy for caries will produce a similar picture and, in great excess, has been known to cause aplastic anemia

Dental Caries. In its earliest stage decay appears as destruction of enamel, indicated first by roughening of the surface, then by a few whitish dull areas surrounded by normal pearly tooth surface and, finally, by cavitation The base and sides of the cavity may be yellow, brown, or black On the biting surface, roughness may not be apparent, discoloration or cavitation being the first sign, on an approximal surface early caries may be detectable only by x-ray A badly decayed tooth shows severe discoloration and destruction Caries may be the result of *improper oral hygiene, faulty nutrition, or systemic disorders dating back* to the formative years In mouth breathers the incidence is high, probably owing to lack of moisture because of evaporation of saliva. In many cases, the cause of decay cannot be explained.

Alveolar Abscess. When caries reaches the tooth pulp, an inflammatory process is set up, indicated by regional discomfort, often constant; pain may be induced or increased by local heat and relieved by cold, or *vice versa* Increase of pain when it is tapped will serve to identify the inflamed tooth. If proper treatment is not initiated, infection spreads into the tooth root and eventually into adjacent bone The result is bone destruction limited to the area surround-

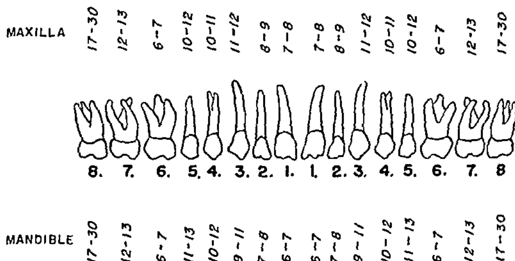


FIG 4 40 Permanent teeth Numbers immediately below teeth refer to names 1, central incisor, 2, lateral incisor, 3, cuspid, 4, first premolar, 5, second premolar; 6, first molar, 7, second molar; 8, third molar Other numbers indicate eruption time in years

teeth or delayed development of permanent ones frequently accounts for improper development of jaws Deformities of the nose or face—deviated septum, high constricted palatal arch, adenoid facies—may be associated with extreme malposition of the teeth. Which is cause and which effect is not clear Faulty breathing and chewing often result

Loosening. Gingivitis or disease of other peridental structures, trauma and the degenerative changes of advancing years cause the teeth to loosen In certain metabolic disturbances such as hyperparathyroidism, loosening may occur as a result of non-suppurative, generalized destruction of alveolar bone

Widened Interdental Spaces. The gradual shifting of a tooth away from its neighbor into the gap left by an extraction (*wandering*) is the most common cause of widening of an interdental space General wandering may be one of the indications of acromegaly, hyperparathyroidism or disturbance of lipoid metabolism such as Gaucher's disease



FIG 4 41 Bilateral unerupted upper third molars impinging on roots of adjacent second molars in a woman age 35 with persistent headache (Courtesy Dr. Ralph E Gove)

the surrounding tissues and usually of the regional lymphnodes. *Any discolored or pulp-treated tooth should be x-rayed to exclude apical abscess.*

Focal infection about the teeth as a possible source of certain constitutional disturbances always deserves consideration but has in some quarters been overemphasized.

Hutchinsonian Teeth. In congenital syphilis, because of developmental hypoplasia, the upper permanent central incisors have a peculiar oval shape and notching of their biting edges. Less often, the first permanent molars are similarly affected (*mulberry molars*).

Teeth Grinding. A habit of oversensitive, nervous children and some adults, teeth grinding does not, as is popularly believed, denote infestation with worms.

THE BUCCAL CAVITY

Exanthematous Diseases. Measles, rubella, scarlet fever, chickenpox, meningococcic infection, smallpox and certain less common exanthematous diseases show eruptions on the buccal mucosa similar in their general characteristics to the cutaneous eruptions The enanthems of scarlet fever, measles and rubella are best seen on and sometimes limited to the soft palate; the others are usually more generally distributed In the rickettsial diseases, enanthem is relatively uncommon.

During the catarrhal stage of measles before onset of the cutaneous eruption, minute bluish-white spots surrounded by red areolae appear in the mouth, chiefly on the cheeks opposite the molar teeth, in the vestibule and, less commonly, on the soft palate. Known as *Koplik's spots,* they frequently supply the key to early diagnosis.

Stomatitis. Inflammation of the buccal mucous membrane has a variety of causes Infection is probably always present, although there may be other underlying factors One or more patches or large areas of mucous membrane show redness, desquamation and probably overlying white deposits of necrotic epithelium When involvement is extensive, salivation is profuse. Circumscribed lesions opposite carious or jagged teeth are common More widespread inflammatory lesions are also found in association with the skin eruption in drug reactions, erythema multiforme and certain other dermatologic disorders Generalized stomatitis may occur with digestive disturbances, in metallic poisoning, and following the use of irritants Pellagra and other vitamin B deficiencies show diffuse inflammatory changes varying from redness to ulceration and sloughing

Administration of Aureomycin, Chloramphenicol or certain other antibiotics, especially when one is given orally and for more than a few days, may be complicated by development of stomatitis, glossitis and cheilosis, varying from a mild inflammatory response to reddening and edema, ulceration, loss of glossal papillae, and cracking, exfoliation and angular fissuring of the lips The picture resembles that seen in pellagra and related deficiency states The mucous membrane may be covered with a whitish, dirty brown or black exudate, prob-



FIG 4 43 Intraoral x rays

A Alveolar absorption due to pyorrhea One third to one half of each tooth root is no longer embedded in bone Arrows indicate level to which bone should reach

B Carious destruction crown of left second premolar with periapical rarefaction of bone (apical abscess) due to infection (Courtesy Dr David Weisberger)

ing the root tip (*apical abscess*), or more diffuse. These changes are detected only by x-ray. By advancing through adjacent bone, the process may reach the labial, lingual, or palatal surface beneath the periosteum and cause severe, constant pain from periosteal elevation or inflammation Here, it may culminate in a subperiosteal abscess discoverable only by x-ray Or it may point into overlying soft tissue, causing pain, swelling and tenderness, this is another form of *gumboil* (see Gingivae) A sinus tract with fistulous opening on the deep aspect of the gingiva may result Sometimes pointing occurs at a more remote site on the cheek or beneath the angle of the jaw, with swelling and inflammation of



FIG 4.44. Hutchinsonian teeth

Purpura. In idiopathic purpura and other blood dyscrasias with bleeding tendencies, petechial hemorrhages and ecchymoses of the buccal mucosa are common. A few petechiae in the mouth may herald severe bleeding.

Blood. Trauma or bleeding from the nose or pharynx may account for the presence of blood or blood stains in the mouth. They are also present in various hemorrhagic diseases and after hemoptysis or hematemesis.

Pigmentation. Brown spots or patches of mucosal pigmentation without elevation or change of texture are not uncommon in any part of the normal mouth, especially in the darker races. Patchy pigmentation is of diagnostic significance in Addison's disease (see Chap. 32). Diffuse dark discoloration is seen in argyria. Small, slightly elevated yellow-brown spots are common on the mucosa of the cheek and lips in many normal persons (*Fordyce's disease*). The lesions are usually few and discrete, but sometimes appear in large numbers, giving a yellow tinge to parts of the mouth. Prolonged use of quinacrine often causes patches of slate blue discoloration, most likely on the hard palate.

Ranula. A thin-walled mucoid cystic swelling appearing on the floor of the mouth near the lingual phrenum, ranula is usually due to obstruction of a sublingual or smaller salivary or mucous duct.

Malformation. High palatal arch is usually associated with developmental malposition of the teeth and alveolar processes or with prolonged obstruction to nasal breathing during childhood from such a cause as hypertrophied adenoids or deviation of the septum.

Torus palatinus is a bony protuberance of the median ridge of the hard palate; it often grows to tumor size. Its contour may be irregular and show multiple crypts. Where it lines the crypts, the mucous membrane is subject to inflammation which attracts the patient's attention to the growth and causes groundless fear of cancer. Torus palatinus often interferes with proper fitting of a denture.



Fig. 4-46. Torus palatinus. (Courtesy Dr. Kurt H. Thoma.)



FIG 4 45 Aphthous stomatitis, left lower buccal sulcus (Courtesy Dr David Weisberger)

ably as a result of superimposed monilial infection, organisms of the *Monilia albicans group* may be seen on stained smear or cultured from the material.

Aphthous Stomatitis. The lesions (*cankers*) begin as circumscribed vesicles, 1–2 mm to 1 cm. in diameter, soon break and become secondarily infected, forming shallow ulcerations, each covered with a whitish exudate and surrounded by a red areola They have a tendency to appear in crops on the tongue and buccal mucosa; they usually disappear within a few days. Some observers believe that they are allergic in origin, others, that they are related to virus infection.

Thrush. Caused by *Monilia albicans*, this is seen at all ages but most frequently in debilitated infants and in older persons in the terminal stages of chronic illness Irregularly distributed over the mucosa are whitish patches surrounded by red areolae; the exudate is more tenacious and adherent than in other types of stomatitis such as Vincent's infection Occasionally areas of redness free of the white membrane will be seen, but search will always reveal one or more patches with the typical appearance

Gangrenous Stomatitis. This rare disease (*noma*) is usually found in a sickly child, especially following some acute infectious disease, particularly measles It starts as a small, red or dusky spot on the inside of the cheek or in the vestibule Ulceration, necrosis and gangrene develop and spread rapidly, soon involving a large part of the cheek and the gingivae. The teeth loosen and fall out, there may be necrosis of the alveolar process of the jaw Externally the cheek is swollen, brawny and, eventually, may show gangrene, sloughing and perforation.

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FIG 4 46 Torus palatinus (Courtesy Dr Kurt H Thoma)



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CLEFT PALATE is a congenital median defect of the hard palate; failure of the palatal processes to unite leaves an abnormal opening between the buccal and nasal cavities.

Other Lesions. Leukoplakia, herpes, Vincent's infection, tuberculosis, syphilis and tumor may affect any part of the buccal mucosa. Their manifestations are similar to those occuring on the tongue, gingivae or pharyngeal mucosa. They must be looked for not only in the easily accessible parts of the mouth but also around the gingival bases and in regions concealed by dental appliances.

THE TONSILS AND PHARYNX

The throat must be examined in a good direct light, preferably daylight. A tongue depressor is pressed firmly but not too forcibly on the middle third of the tongue and, to elevate the soft palate and uvula, the patient is asked to enunciate a prolonged "Ah h-h." Placing the blade too far back or pressing too hard or suddenly will cause gagging Where the reflex is particularly active, gagging can be avoided and a satisfactory view obtained by substituting, for the conventional "Ah-h-h," a series of short gasping breaths through the mouth When, as sometimes happens, the patient cannot relax his tongue sufficiently to permit a good view, the examiner must rely on one or two brief glimpses obtained by deliberately forcing him to gag Structures to be observed are.

1. Soft palate and uvula.
2. Palatine arches
3. Tonsils
4. Posterior pharyngeal wall.

Diseased states, if involving one part, usually affect all, but distribution of lesions has an important bearing on the differential diagnosis

Exanthematous Diseases. As noted above, the enanthems of measles, rubella and scarlet fever are best seen on and sometimes limited to the soft palate Those of chickenpox, smallpox and others may appear on the soft palate but are just as common elsewhere on the buccal mucosa In addition to the palatal eruption scarlet fever shows marked redness and edema of the faucial ring similar to that of acute septic inflammation

Acute Pharyngitis. Congestion and edema cause the mucous membrane to appear red, shiny and swollen The uvula is swollen and its tip transparent Some observers believe that if elongation of the uvula brings it into contact with the tongue, it may excite persistent cough, gagging or swallowing, others doubt this

Acute Tonsillitis. The tonsils are red and swollen, in more severe cases they are studded with small, white or yellowish patches of exudate extending from within the crypts Such patches may extend and coalesce, giving the appearance of a membrane

Acute Septic Inflammation. The structures of the throat are bright, fiery red, greatly swollen and usually partly covered by a grayish-white, filmy or veil-like exudate In contrast to the dense, adherent membrane of diphtheria, this



Fig 4 47 Early herpetic lesions on hard palate (Courtesy Dr David Weisberger)



Fig 4 48 Extensive carcinoma of buccal mucosa, left side (Courtesy Dr David Weisberger)

to be visible. In such cases, digital examination may reveal bulging and, sometimes, fluctuation, usually to one side of midline A lateral x-ray of the neck will show widening of the soft-tissue shadow between the pharynx and vertebral bodies. Extension of infection from tuberculous cervical lymphnodes or cervical Potts' disease may cause chronic retropharyngeal abscess.

Vincent's Infection. Although the gingival or buccal mucosa is usually involved first, the infection sometimes starts on one tonsil, appearing as a localized ulceration covered by a grayish slough. It is to be suspected in any case of unilateral tonsillar inflammation. When the faucial ring is involved, the disease may be confused with diphtheria and ulcerative lesions such as syphilis and agranulocytosis.

Agranulocytosis. Some cases show at onset a few or many pin-head-sized superficial yellowish ulcerations with red, inflammation margins; they begin on any part of the faucial ring, buccal mucosa or gingivae. Other cases start with redness and tremendous edema of the pharyngeal wall and tonsils The infection spreads rapidly to other parts of the mouth. Necrosis apears early; the necrotic areas are covered by a dirty-gray, easily removable slough. The process burrows deeply, death from hemorrhage due to erosion of an artery has been known to occur. *Edema is always a prominent feature and, if unilateral, may in the earlier stages be so marked as to lead to an incorrect diagnosis of peritonsillar abscess* The clinical appearance is quite similar to that of Vincent's infection, in fact, the organisms of the latter may be found in the lesions but in smaller numbers Diagnosis is confirmed by examination of the blood or bone marrow. This serious disturbance must be watched for during therapy with certain agents, especially arsenicals, gold salts, sulfonamides, the thiouracil group, aminopyrine, chloramphenacol, marrow-suppressive drugs used in the treatment of leukemia, many of the newer so-called tranquilizers, and radiation

Infectious Mononucleosis. Over 50 per cent of cases of infectious mononucleosis show some inflammation of the throat There is injection of the fauces with swelling of the lymphoid tissue (*granular pharyngitis*). Mild follicular tonsillitis may appear. Occasionally membranous angina suggesting diphtheria or agranulocytosis occurs

Chronic Pharyngitis. Some cases are characterized by congestion of the uvula, soft palate and posterior pharyngeal wall, perhaps with nodules of hypertrophied lymphoid tissue. Others show a glazed, dry mucous membrane Chronic pharyngitis is common in heavy smokers and drinkers and in persons exposed to excessive dust, and may be secondary to chronic adenoid or sinus infection or to atrophic rhinitis.

Chronic Tonsillitis. Frequent attacks of severe or mild acute tonsillitis or persistent low-grade sore throat indicate chronic tonsillar infection It should be suspected in patients with systemic manifestations of low-grade infection or toxic absorption occurring even without repeated sore throat, especially when there is a history of severe tonsillitis or peritonsillar abscess in the past Localized redness of the tonsils and pillars, perhaps enlargement of the subtonsillar



FIG 4 49 Faucial diphtheria Arrows point to dirty gray membrane on tonsils and soft palate (Courtesy Dr Louis Weinstein, Chief, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

exudate as well as that sometimes seen in acute tonsillitis can be easily wiped off

Diphtheria. Typical cases show a duller, more purplish redness of the mucosa, but most characteristic is the dull, grayish-white membrane which may cover only a small area of the tonsil or pharynx or involve as much as both tonsils and pillars, the uvula, soft palate, and part of the hard palate The membrane is slightly raised above the surface, firmly attached, and—if removed—leaves a raw, bleeding surface beneath *The area of redness and inflammation in diphtheria characteristically spreads only a short distance beyond the border of the membrane,* in scarlet fever and septic inflammation the opposite is true

Peritonsillar Abscess. A complication of acute tonsillitis, an abscess develops in the capsule between the tonsil and constrictor muscles of the pharynx Pain is marked Inflammation of regional muscles causes dysphagia and partial trismus which may be severe enough to interfere with examination. Local redness is present, swelling on the involved side is so marked as usually to hide the tonsil and displace the median raphe and uvula The regional cervical lymphnodes are swollen and tender, inflammation of the deep cervical muscles may cause the head to be held rigidly and perhaps inclined toward the affected side

Retropharyngeal Abscess. Essentially a disease of infancy and early childhood, retropharyngeal abscess forms between the posterior pharyngeal wall and the prevertebral fascia. It is caused by suppuration of a posterior pharyngeal lymphnode secondary to acute pharyngitis The child is often too young to complain of pain but unexplained fever, loss of appetite or difficulty in swallowing should suggest the diagnosis If the airway becomes narrowed, irritative cough and perhaps stridulous breathing develop Swelling of the posterior pharyngeal wall may be seen, but often the abscess is too high or too low

most normal persons will be elevation of the palate with gagging or cough, and perhaps simultaneous tearing of the eyes and contraction of facial and abdominal muscles (*gag reflex*). Occasionally in the normal and frequently in the high strung or apprehensive patient this reaction will be initiated by touching virtually any part of the mouth or occur even before the stimulus is actually applied. A diminished or absent reflex may be encountered in normal persons and occasionally in hysterics. It can also be a reflection of palatal paralysis due to a lesion of the tenth nerve as in bulbar poliomyelitis, diphtheria, progressive bulbar palsy or a vascular lesion of the medulla, or rarely of sensory impairment due to a lesion involving the ninth nerve or its pathways. With a motor disturbance, since sensation is intact, the stimulus may still cause tearing of the eyes or contraction of abdominal muscles even though the palate does not move.

Palatal Paralysis. Partial or total paralysis of the palatal muscles, which are supplied by the tenth cranial nerves, is manifested by nasal speech, regurgitation of liquids through the nose during swallowing and visibly impaired movement of the uvula or soft palate With slight paresis there may be only disturbance of speech without detectable impairment of movement. When paresis is severe and bilateral, the palate does not rise on phonation; when it is unilateral the palate fails to rise in midline, but is pulled by the unaffected muscles away from the affected side. The usual causes are.

1. Postdiphtheric paralysis
2. Poliomyelitis with bulbar involvement
3. Progressive bulbar paralysis In all but a few advanced cases the palate does not fail to rise, but functional derangement is indicated by the nasal voice
4. Idiopathic polyneuropathy.
5. Pseudobulbar paralysis, due most likely to thrombosis or embolism of the basilar artery or to bilateral apoplexy.
6. Tumor or infarct of the medulla.
7. Neurosyphilis
8. Myasthenia gravis.
9. Botulism

THE NASOPHARYNX

In trained hands, the electric nasopharyngoscope introduced through the nose provides the best view of the nasopharynx For the less experienced, examination by the nasopharyngeal mirror and reflected light must suffice. While the midportion of the tongue is held firmly down with a depressor, the mirror is slipped behind the soft palate. Because of the gag reflex, care must be taken to avoid touching the uvula, tongue and posterior pharyngeal wall The mirror is then rotated so as to direct its reflecting surface toward the patient's forehead, thus bringing the nasopharynx into view When the gag reflex is exaggerated, the uvula, soft palate and surrounding structures may be sprayed with a mild local anesthetic

One should look first for the posterior edge of the septum, which can be seen

lymphnodes and excessive caseous material in the crypts are the signs to be looked for Purulent material can sometimes be expressed from the crypts by pressing on the anterior pillar. An enlarged, infected tonsil may be embedded behind the anterior pillar and *appear small and innocuous; forced gagging or* external pressure on the neck below the angle of the jaw will bring it into view

Syphilis. CHANCRE should be suspected in cases of protracted enlargement and ulceration of one tonsil.

SECONDARY *syphilis may simulate simple*, acute pharyngitis or cause mucous patches on the tonsils and pillars as well as elsewhere in the mouth.

TERTIARY syphilis of the tonsils, palate, or soft palate is marked by a deep, "punched-out," chronic ulcer with a greenish-yellow base; it is relatively painless. *Perforation of the soft palate or adhesion to the back of the pharynx almost always means syphilis.*

Tuberculosis. Pharyngeal tuberculosis, always secondary to active pulmonary tuberculosis, is marked by irregular patches of chronic ulceration on the wall, occasionally one may see, in the tonsillar region, tubercles resembling canker sores but more persistent In contrast to syphilis, tuberculous lesions are painful and tender.

Angioneurotic Edema. Painless swelling, with translucent pallor of the soft palate, fauces or uvula appears with characteristic suddenness, disappearing again after some hours It may be associated with allergic phenomena elsewhere.

Benign Tumor. Papilloma, adenoma, fibroma, hemangioma and other benign neoplasms may occur in the pharynx, but are rare The tumor is circumscribed, fairly firm, and either sessile or pedunculated

Malignant Disease. CARCINOMA may involve any part of the pharynx, but is more frequent on the lower lateral wall It appears first as a painless, hard, nodular swelling which later ulcerates and becomes painful Sometimes it is not visible but can be felt on digital examination

LYMPHOMA may involve one or both tonsils primarily or as part of widespread disease of the lymphatic system The affected tonsil will appear as a bulging, lobulated, spongy mass retaining fairly normal color Later, necrosis with secondary infection may develop The soft palate and uvula may bulge downward and forward, betraying the presence of tumor growth in the posterior nasopharynx In rare instances, the bilateral tumor meets in the midline, causing obstruction to swallowing and, perhaps, to respiration

LYMPHOSARCOMA manifests itself as fiery red, painful, tender swelling of the pharynx or tonsil which soon develops into an ulcerating tumor. No other lesion of the tonsils, except abscess, grows so rapidly and invades surrounding tissue so extensively. In abscess, pain, fever and constitutional symptoms are greater

Malignant tumor of the parotid gland, by extending medially, may cause bulging of the pharyngeal wall or tonsil.

Pharyngeal Reflex. If, with a tongue depressor or comparable object, one touches the dorsum of the tongue or any part of the faucial ring, the response in

FIG 4 50 Fibroma of larynx extending upward into pharynx. (Courtesy Dr David Weisberger)

but inclined slightly forward from the waist With his left hand, the examiner holds the patient's tongue forward and with his right places the mirror toward the back of the pharynx, avoiding contact with the tongue, hard palate, fauces and posterior pharyngeal wall. With the back of the mirror resting against the anterior surface of the uvula, the reflecting surface is tipped downward *until the larynx is brought into view. In patients who gag easily it is advisable* to spray the throat with a mild anesthetic.

The anterior portion of the larynx appears in the upper part of the mirror, the posterior or arytenoid region is seen below Between them, converging vertically from the right and left to the anterior commissure, are seen the vocal cords The color and condition of the mucous membrane, cords, arytenoids,

FIG 4 51 Mirror view of normal larynx

in the center of the field as a pink ridge, becoming thicker and merging at its upper end with the roof of the nasopharynx. The mucosa is normally pink and smooth, at the apex of the cavity may be seen the mucosal folds of adenoid tissue Flanking the septum are the posterior nares through which appear on each side the smooth, rounded, pale superior turbinates, and the posterior aspects of the middle and lower turbinates Turning the mirror to either side will bring into view the opening of the Eustachian tube and the cartilaginous ridge (*torus tubaris*) curving above and behind it Beyond the ridge lies a depression known as the *fossa of Rosenmuller*

Digital examination is often helpful, especially in children, in whom use of the pharyngeal mirror or nasopharyngoscope is difficult. With a gag between the patient's teeth to prevent biting, the examiner inserts his forefinger behind the edge of the soft palate and up into the nasopharynx, rapidly palpating the roof, the Eustachian orifices and the posterior ends of the lower turbinates Hypertrophied adenoid tissue, enlargement of the posterior part of a lower turbinate, perhaps swelling of a Eustachian orifice or an inflammatory or neoplastic mass may be felt.

Acute Nasopharyngitis. The mucous membrane is swollen, red and secretes a watery or thin mucoid discharge

Hypertrophied Adenoid Tissue. Resulting from repeated infection, hypertrophied adenoid tissue appears as an irregular, pink, edematous mass occupying a large part of the nasopharynx. On digital examination one can feel an irregular, boggy mass almost filling the space. Mouth-breathing, prolonged and frequent head colds, recurring attacks of otitis media and, often, defective hearing result

Purulent Secretion. Pus or mucopus in the nasopharynx usually indicates sinus infection; if seen in one or another meatus, involvement of the corresponding sinus is likely

Tumors. POLYPS, usually arising in the antrum, less often in the other nasal sinuses, appear in the nasopharynx as pallid, shiny, smooth, rounded masses partly or wholly occluding the posterior nares

THORNWALDT'S DISEASE, also called cystic bursitis and due to a developmental variant of the pharyngeal mucosa, appears in the nasopharynx as a smooth, round, red mass which may be mistaken for adenoid growth It is differentiated by its regularity of outline, retained secretion, palpable fluctuation, and sometimes expressible mucopurulent material.

FIBROMA is a firm, circumscribed tumor.

MALIGNANT TUMOR usually appears as an ulcerated, irregular mass which can be identified only by biopsy

THE LARYNX

Hoarseness, aphonia, and croupy cough direct attention to the larynx Direct laryngoscopy is the most effective method of examination but requires special training For ordinary purposes, the indirect method, performed with a mirror and reflected light, is sufficient. The patient should sit well back in the chair

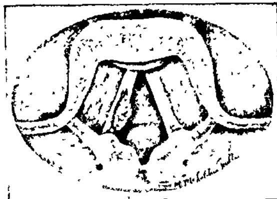


Fig 4 52 Early carcinoma of right vocal cord

Cancer may also extend downward from the pharynx and encroach upon the larynx. As is true elsewhere, the differential diagnosis between cancer, tuberculosis, and syphilis cannot be made from the appearance of the lesion. History, general clinical picture, serologic tests and often biopsy are necessary.

Paralysis of Vocal Cords. One cannot always rely on a single examination Apprehension or tenseness may cause spasm of the cords that is easy to confuse with paralysis, making it advisable to repeat the examination until the laryngeal irritability is reduced. Paralysis of the cords may be partial or complete, unilateral or bilateral. Partial paralysis is usually asymptomatic Complete paralysis of one cord causes hoarseness, of both cords, aphonia. Unilateral paralysis is usually due to pressure on or damage to a recurrent laryngeal nerve from some intrathoracic lesion such as mediastinal tumor, aneurysm or dilated left auricle, or from some cervical lesion such as goiter, tumor or lymphnode enlargement Bilateral paralysis may result from one of the above but is more likely due to postdiphtheritic neuritis or a disorder affecting the tenth nerve nuclei in the medulla, such as anterior poliomyelitis or progressive bulbar

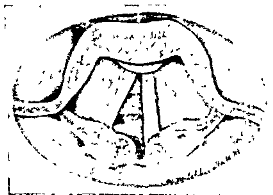


Fig 4 53 Paralysis of left vocal cord due to impaired function of left recurrent laryngeal nerve. Cord is in midline instead of being deviated toward left

and the rest of the epiglottis are noted. Membranes, ulcerations, and growths should be looked for.

The true cords *are normally pearly gray and shiny. Their movements can* be tested by asking the patient to enunciate a prolonged "Eh-h-h" They come close together and are taut on phonation. On inspiration they relax and the arytenoids fall laterally, revealing a view of the wall of the trachea below. Occasionally the upper rings of the trachea can be seen.

Laryngitis. Acute laryngitis produces redness, congestion and edema of the entire larynx with resultant hoarseness and croupy cough. In severe streptococcic infection, edema is pronounced.

Chronic laryngitis may be associated with chronic infection higher in the respiratory tract, especially sinusitis, or with chronic bronchopulmonary infection and is common without infection in public speakers, singers and others who strain their voices Injection and thickening of the cords and adjacent structures are observed

Foreign Body. *This must always be looked for in any obscure case of hoarse*ness or harassing cough, particularly in children and psychotics

Diphtheria. Redness, edema and a dirty gray membrane are present, the picture resembles that of faucial diphtheria

Tuberculosis. Almost always occurring as a complication of pulmonary tuberculosis, laryngeal tuberculosis is usually seen first in the posterior third. In the earlier stages the epiglottis is pale As the process continues, one sees interarytenoid thickening, then possibly a slight superficial ulceration of the posterior third of the larynx The final stage shows tremendous edema with ulceration throughout, involving mostly the epiglottis and arytenoids Talking and swallowing are extremely painful

Syphilis. Chancre is rare In secondary syphilis, hyperemia of the mucosa and often edema, especially of the aryepiglottic folds, are observed, mucous patches may be present Late syphilis is characterized by smooth swellings on any part of the mucosa, especially the epiglottis and ventricular folds One or more may progress to ulceration, the floor of an ulcer will be dirty-drab and the edge overhanging

Tetany. Attacks of laryngeal spasm cause partial respiratory obstruction indicated by a high-pitched crowing sound with each inspiration *(laryngismus stridulus).* During a severe bout, periods of apnea, with marked respiratory distress and cyanosis, occur.

Tumors. Papilloma is usually seen attached to a true cord around the anterior third of the glottis, freely movable, pale red, and with typical cauliflower-like papillomatous appearance

Polyp is soft and pallid, varies in size and shape and can be either sessile or pedunculated

Vocal nodules, which appear as small excrescences on the cord margins, are seen in singers and others who strain their voices.

Carcinoma usually begins as a papillary thickening of one cord but soon ulcerates, showing a dirty-white slough. Later fixation of the cord develops.

5

THE NECK

POSITION AND MOTION

Abnormal position of the head and neck can be seen at a glance It is usually accompanied by local tenderness and limitation of motion. One determines active mobility by noting the degree to which the patient can himself rotate his head and bend it forward, backward and to each side; passive motion is determined by noting how far the examiner can rotate or bend the head in various directions before encountering resistance Caution must be exercised when disease or injury of the vertebral column is suspected, sudden or vigorous manipulation may cause fracture or dislocation with resultant damage to the spinal cord (For diagrams of tests for mobility see Chapter 35)

Stiffness. Stiff neck usually accompanied by local pain and limitation of motion in one or more directions, results from the causes listed below. Depending on the etiology, it is transient or permanent.

MUSCULAR SPASM. This is sometimes unexplainable but more likely due to one of the following:

1 Exposure, especially to a draft.

2 Strain, acute or chronic.

3 Compensation for curvature elsewhere

4. Abnormal position of the head due to faulty vision or occupational stress.

5 Disease of the soft tissues, such as inflamed or enlarged lymphnodes, cellulitis, abscess, tumor, or trichiniasis involving the cervical muscles.

6 Meningeal irritation, as in meningitis, anterior poliomyelitis, subarachnoid hemorrhage, and posterior fossa tumor Here only anterior-posterior movements are affected The slight stiffness of early involvement can be best brought out by lifting the patient's head from the table while at the same time holding his thighs down, resistance will be encountered before flexion of the head is complete. Occasionally mild stiffness will be more evident if the patient attempts to raise his head voluntarily. In the equivocal case the head should be passively raised without pressure on the thighs, meningeal irritation may then be indicated by involuntary flexion of the thighs and legs (*Brudzinski's neck sign*) When severe, rigidity of the neck and back may be so marked as to cause hyperextension (*opisthotonos*)

7 Parkinsonian syndrome, and certain other neurologic disturbances

8 Tetanus.

paralysis. It also occurs in botulism In hysterical aphonia laryngoscopic examination may reveal pseudoparalysis of the cords difficult to distinguish from true paralysis

DYSPHAGIA

The manifestations of a swallowing disorder may be entirely subjective. The patient will complain of mild choking, regurgitation, or food or fluid seeming to "stick" in the throat or further down If pain is severe or obstruction pronounced, there will be obvious signs of effort, distress or choking Paralysis of any of the muscles of deglutition will cause nasal voice, regurgitation of fluids through the nose and perhaps drooling and spasms of coughing Common causes of dysphagia are:

Inflammatory Lesion in Mouth. In severe stomatitis, tonsillitis, tuberculosis, cancer or other inflammatory or neoplastic disease of the mouth, pharynx or neighboring structure, dysphagia may be due entirely to pain but edema or interference with the swallowing mechanism may contribute.

Mechanical Defect in Mouth. Such disturbances as cleft palate, angioneurotic edema, fixation of the tongue by malignant infiltration and syphilitic pharyngeal stenosis may cause dysphagia by interfering with the normal passage of food or fluid.

Esophageal Disorders. Dysphagia may be caused by pain, obstruction, or both in such disturbances as esophagitis, erosion or ulceration, foreign body, neoplasm, diverticulum, achalasia, constriction from corrosive burn or other injury, *angioneurotic edema, or pressure from without as by goiter, retropharyngeal* abscess, aneurysm or mediastinal tumor.

Nerve or Muscle Dysfunction. Impairment of activity of one or more muscles concerned with deglutition will produce dysphagia, its degree depending on the muscles affected and the extent of damage The most common causes are:

1. Disease affecting the brain stem, such as bulbar poliomyelitis, progressive bulbar palsy, tumor, infarction, syphilis, tetanus, hydrophobia, or botulism
2. Peripheral neuropathy, as in postdiphtheritic paralysis and severe idiopathic polyneuropathy
3. Muscular weakness, as in extreme wasting diseases and myasthenia gravis
4. Hysteria

with the physician standing behind the seated patient. With firm but not too rigid hands and fingers, he should, for the sake of comparison, feel the two sides simultaneously. Nodes which might be concealed by a sternomastoid muscle are detected in the following manner: The patient's head is tilted toward the homolateral side to relax the muscle. If the thumb is now pressed deeply behind it, the nodes will be projected forward toward the anterior muscular border, where they can be felt by the fingers For the submaxillary area, bimanual palpation is required. The gloved finger of one hand is placed on the floor of the mouth medial to the ramus of the jaw and pressed downward and outward, thus projecting the contents of the area against the fingers of the other had placed externally beneath the ramus. The submental region should be similarly palpated with the fingers of one hand pressing downward and forward from beneath the alveolar process of the incisor teeth, and the fingers of the other hand held beneath the chin. Enlarged supraclavicular nodes must be searched for with the patient upright; if he is recumbent they may descend below the clavicle and be overlooked. A swelling of the neck thought to be an enlarged lymphnode may be a swollen salivary gland, an abscess, a tumor arising from the spine, carotid body, or other cervical structure, a small cyst or a carotid aneurysm The so-called island of aberrant thyroid tissue sometimes found in the neck or its vicinity is thought by most observers to be a metastatic lymphnode from thyroid carcinoma

Pathologic examination in practically all cases of chronic and in many cases of acute swelling of cervical nodes or lesions simulating such nodes is imperative Diagnosis based on clinical findings alone is risky The commonest causes of lymphnode enlargement are.

Regional Inflammatory Process. Acute or chronic infection of the mouth, throat or other part of the face or head such as tonsillitis, diphtheria, alveolar abscess, Vincent's infection and stomatitis is usually accompanied by secondary lymphnode involvement. Swollen nodes in the postauricular and occipital areas and in the posterior triangle, notably in children, strongly suggest pediculosis capitis

Rubella. Enlargement and tenderness of the *posterior cervical or occipital* nodes without involvement of others occurs early in German measles This finding, when present along with mild coryza, especially in an epidemic, may make diagnosis possible before the eruption appears

Infectious Mononucleosis. Occurring chiefly in young adults and children, this acute infection produces fever, swelling and tenderness of the cervical lymphnodes and almost always the axillary, inguinal and other nodes Faucial inflammation is common at onset Occasionally there may be severe stomatitis The spleen is often palpable Diagnosis depends on the general clinical findings and the blood picture; it may be confirmed by the demonstration of high titer of heterophile antibodies in the blood serum Lymphnode enlargement may persist for weeks or a few months after recovery from the acute phase; convalescence is often prolonged

Granulomatous Disease. TUBERCULOUS enlargement *usually* appears first in

9. Congenital wry neck (*torticollis*).

DISEASE OR INJURY OF VERTEBRAE. The most common are:

1. Arthritis
2. Tumor
3. Tuberculosis, or some other infectious and destructive process.
4. Dislocation or fracture of a vertebra or an intervertebral disc

A sensation of tenseness, tightness of stiffness in the back of the neck without limitation of motion or other objective sign is common in high-strung persons, *especially during periods of fatigue or emotional stress*

Muscular Weakness. Difficulty in holding up the head may occur in:

1. Severe wasting disease.
2. Chorea
3. Myasthenia gravis.
4. Poliomyelitis, progressive muscular atrophy, and certain rarer neurologic disorders

THE SKIN AND SUBCUTANEOUS TISSUE

Abscess. ACUTE abscess on the side or front of the neck is usually the result of suppuration of lymphnodes secondary to infection in the mouth, throat or other adjacent structure. The nape is a favorite site for furuncles and carbuncles The primary lesion of anthrax is likely to appear on the neck

CHRONIC abscess, usually associated with draining sinuses, occurs in lymphnode *tuberculosis, actinomycosis, coccidioidomycosis, other granulomatous* infections and secondary to break-down of malignant lymphnodopathy Tuberculosis of the upper cervical vertebrae may be complicated by draining sinuses *in the neck, in adults irregular, multiple scars suggest lymphnode tuberculosis* in childhood. In males chronic folliculitis of the beard area is common as a result of repeated infection, most likely from a shaving brush

Edema. Inflammatory disturbances originating in the mouth, throat, salivary glands and other regional structures can cause cervical edema, the lymphnodes are usually involved also The neck and face are swollen in superior or anterior mediastinal syndrome

*LUDWIG'S ANGINA refers to an acute inflammatory process involving the sub*lingual and submaxillary spaces. Most commonly resulting from peridental infection or tooth extraction, it is characterized by edema, tenderness and elevation of the floor of the mouth, edema of the tongue, and tenderness, swelling and redness of the upper anterior portion of the neck, especially about the submaxillary gland.

ENLARGED LYMPHNODES

Examination of the neck for enlarged lymphnodes should be systematically performed Corresponding regions—the preauricular, mastoid and suboccipital areas, *the anterior and posterior triangles*, and the supra- and infraclavicular fossae—should be inspected and palpated in turn Palapation is best performed

Intra abdominal or intrathoracic nodes may be affected without evidence of peripheral disease.

Leukemia. Lymphocytic and often other forms of leukemia cause slow, painless, non-tender, discrete enlargement of nodes. They do not become fixed or fluctuant. As in Hodgkin's disease, involvement may be limited to the neck or be generalized. Diagnosis is established by examination of the blood, bone marrow or sometimes an excised node.

Carcinoma. Usually secondary to cancer of the lip, tongue, mouth, esophagus, sinus, thyroid gland or other structure of the head or neck, the nodes are discrete, hard and of varying size Fewer nodes are involved than in the diseases described above. These metastases are sometimes evident earlier than the primary lesion; careful search may fail to reveal their source until late in the disease Malignant tumor of the stomach, lung, ovary, uterus or other distant organ may cause metastatic enlargement of one or more nodes above or just below the clavicle usually on the left. Such *sentinel nodes* should be searched for in any obscure case where intra-abdominal or intrathoracic malignant disease is suspected. Metastatic involvement of the supraclavicular group is a late complication of cancer of the breast. In the presence of thyroid enlargement, one or more palpable lymphnodes just off midline above the isthmus are highly suggestive of carcinoma or thyroiditis (*Delphian nodes*)

THE SALIVARY GLANDS

The Parotids

Normally, none of the salivary glands can be seen or felt Swelling of a parotid is visible and palpable under the angle of the jaw and behind and in front of the lower part of the ear Limitation by its sheath usually prevents extension beyond the level of the zygoma. The lower edge of the swollen gland may be well-demarcated, but elsewhere the swelling merges into surrounding tissue

Systemic diseases which affect one set of salivary glands often, but not always, involve the others.

Epidemic Parotitis (Mumps). One or both parotid glands become swollen, tender and often painful Inspection of the buccal mucosa adjacent to the second molar tooth on the affected side may disclose swelling and redness of the papilla at the outlet of Stensen's duct. Systemic symptoms may be mild or severe One or both submaxillaries or rarely the sublinguals may also be involved, sometimes without the parotids Involvement of the pancreas or one or both testes or ovaries may occur, systemic response is then more severe

Acute Parotitis. Inadequate drainage due to a calculus or unexplained atresia of Stensen's duct will produce pain, tenderness and swelling of the gland Recurrent attacks are usually due to a calculus, occasionally to cystic degeneration of the main duct radicals within the gland. Diagnosis can sometimes be established by a plain x-ray film or films taken after injection of contrast





Fig 5 1 Fig 5 2

Fig 5 1 Tuberculous lymphnoditis with abscess
Fig 5 2 Swelling of neck due to enlarged nodes in lymphoma

the carotid group, just behind and below the angle of the jaw, next in the submaxillary, and then in the supraclavicular group The nodes at first are fairly firm, though they do not have the hard, resistant feel of cancer Later there is a tendency toward softness within the firmness and, eventually, definite softening and fluctuation. When multiple nodes are involved, all stages of the process may be present The nodes feel matted together and may be adherent to the skin, sternomastoid muscle, or deeper cervical tissues Longstanding cases may show draining or healed sinuses

Syphilis shows enlarged and usually somewhat soft regional nodes in chancre of the mouth or face Slight, firm, non-tender enlargement, usually of the occipital, postcervical and postauricular nodes occurs in secondary syphilis as part of the general lymphnodopathy Gumma of a lymphnode may occur in late syphilis, it cannot be distinguished from a tuberculous lesion by local examination.

Sarcoid and other granulomatous diseases give a picture simulating tuberculosis with or without necrosis and sinus formation, depending on the etiologic agent

Malignant Lymphoma. Hodgkin's disease and other forms of lymphoma are characterized by chronic, non-tender, firm, discrete, multiple lymphnode enlargement. In the late stages, the nodes become greatly enlarged, fused and adherent to each other; occasionally they extend through the skin and suppurate The neck is the most common site of peripheral involvement; the axillary, inguinal and other peripheral nodes may or may not be affected, but one rarely encounters diseased nodes in these areas without cervical involvement.

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material *If suppuration develops, it may be possible by stroking the cheek along the course of Stensen's duct to express pus from the meatus.*

Acute inflammation of a parotid gland can also occur from extension of an infectious process in the mouth, during some acute systemic infection or chronic debilitating disease, and following a serious abdominal operation, especially in an older person with cancer Dehydration may be a factor. The process is usually unilateral; if both glands are involved, the second becomes affected as the inflammation in the first has reached or just passed its peak. Suppuration is likely

Acute swelling of the parotids, usually with similar swelling of the other glands, is one of the signs of intoxication by thiouracil or an allied drug

Chronic Parotitis. Persistent low-grade swelling, pain and tenderness may result from obstruction of Stensen's duct or cystic enlargement of its radicals Chronic bilateral enlargement of the parotid and lacrimal, with perhaps enlargement of other salivary glands is seen in Mikulicz's syndrome (see Chap 4) The glands are firm, painless, non-tender and non-adherent to adjacent structures. In uveoparotid fever, thought to be a form of sarcoid, there are recurrent bouts of fever, swelling and tenderness of the parotids and, due to the uveitis, blurring of vision, photophobia and lacrimation

Tumor. Visible or palpable non-tender fulness or swelling is the first indication The type of neoplasm can often be determined only by pathologic examination

MIXED TUMOR, the most common form, likely to appear in the third or fourth decade, is firm, sharply defined, movable and slow-growing. It tends to recur *following removal unless a wide resection is performed* Although mixed tumor

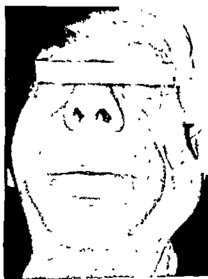


FIG 5 3 Local swelling of face and neck due to mixed tumor of parotid gland (Courtesy Dr. David Weisberger)

is usually regarded as benign, it appears to be well-established that some cases of carcinoma arise from it

CARCINOMA, more likely to appear in the seventh or eighth decade, is hard, fixed, less sharply demarcated than mixed tumor, and grows more rapidly. Metastatic enlargement of regional cervical lymphnodes and facial palsy secondary to involvement of the seventh nerve are common and of some diagnostic significance.

SALIVARY TUMOR is occasionally found in one of the other glands or in an aberrant location, especially the cheek, palate, or lip

THE SUBMAXILLARIES

When enlarged, a submaxillary gland appears as a palpable, fairly circumscribed, sometimes visible swelling beneath the mandible Unilateral swelling may occur in mumps or with calculus in Wharton's duct. Bilateral enlargement may occur in mumps, Mikulicz's syndrome, and other systemic disturbances affecting the salivary group Mixed tumor or carcinoma is occasionally encountered

THE SUBLINGUALS

An enlarged sublingual gland is felt as a small swelling on the floor of the mouth to one side of the phrenum Swelling may result from cystic dilatation of the duct (*ranula*), or in any of the disturbances described above

THE THYROID GLAND

One inspects the thyroid region particularly for enlargement or abnormal shape of the gland, for nodules, pulsations, and for displacement of the trachea Ordinarily the gland is not visible but in a thin person it is sometimes sufficiently prominent, especially in the region of the isthmus, to be confused with enlargement Occasionally a small pad of subcutaneous fat above the sternal notch may be mistaken for an enlarged or nodular gland, it is softer, smaller, more freely movable than the gland and, in contrast to the latter, can to some extent be lifted from underlying structures and does not rise and fall with deglutition With these two exceptions, swelling or prominence in the suprasternal area is most likely due to nodular or diffuse enlargement of the thyroid gland Asymmetry, enlargement, or a nodule which might otherwise be overlooked, can sometimes be seen by having the patient hyperextend the head and swallow

Palpation is usually performed by standing behind the seated patient and feeling with the fingers, firmly but not rigidly encompassing the anterior and lateral portions of the lower neck lying between the sternomastoid muscles Some observers perfer to examine from the side with one hand palpating the thyroid region and the other steadying the head from behind In either case, one should palpate, in turn, with the head in the normal position, flexed and hyperextended, first with the muscles at rest, then during the act of swallowing.

The latter procedure is expedited by having the patient drink successive mouthfuls of water; while he is holding the glass, there is also a good opportunity to look for tremor of the hand, which is an important feature of thyrotoxicosis. After this routine, each lateral lobe should be examined, while it has been displaced laterally, by pushing its counterpart toward the midline. One should also feel on each side of midline just above the isthmus for an enlarged lymphnode; if one is found in the presence of thyroid enlargement or nodularity, malignant disease or thyroiditis is to be suspected.

The thyroid gland rises and falls with the larynx during *deglutition*; other cervical structures do not Ordinarily none of the gland is palpable, except perhaps for the isthmus, which is sometimes felt as a soft prominence overlying the trachea below the cricoid cartilage If other parts of the gland are palpable, enlargement is indicated, except when the neck is unusually thin. The pyramidal lobe is felt for by sliding the examining finger back and forth across midline just above the isthmus, it may be felt as a upward finger-like projection If palpable, it is probably abnormal If, with the head hyperextended, the lower edge of an enlarged gland cannot be demarcated, it probably extends beneath the sternum Light palpation may reveal a vascular thrill associated with an audible bruit.

In addition to determining the size of the gland, one feels also for nodules: their size, shape, location, consistency, mobility, and sharpness of outline. Nodules may be single or multiple, hard or soft, poorly or well defined

Auscultation may disclose a bruit indicative of increased vascularity and occurring only in hyperplastic goiter Bruit is a fairly continuous humming sound accentuated during cardiac systole and heard diffusely over the gland. It must be distinguished from the systolic sound sometimes audible over the arteries of the neck, and from the physiologic venous hum heard just above the clavicle The latter may be obliterated by pressing on the jugular vein (see Chap 12)

Transillumination may occasionally be helpful in distinguishing between a cyst and a solid nodule.

An appreciably enlarged thyroid may produce pressure symptoms such as tightness or drawing in the neck, or difficulty in breathing or swallowing

Colloid Goiter. This is most likely to be found in adolescent females and in others during pregnancy The gland is diffusely enlarged, soft, poorly demarcated, non-tender and without bruit Thyroid function is normal or slightly diminished. The disturbance may regress spontaneously or develop into nodular goiter.

Hyperplastic Goiter. The gland is firm, elastic, and quite clearly demarcated; a bruit is often audible. This is *the form of goiter most commonly associated* with thyrotoxicosis The manifestations of the latter, not all of which are necessarily present, are. loss of weight despite increased appetite, emotional instability, excessive *sweating, diarrhea, palpitation and other* indications of cardiac embarrassment, exophthalmos and other ocular signs (see Chap 4), fine tremor of the fingers and hands, tachycardia, and elevated basal metabolic





Fig 5 4 Fig 5 5

Fig 5 4 Simple colloid goiter

Fig 5 5 Thyrotoxicosis Pronounced exophthalmus with moderate degree of diffuse thyroid enlargement

rate The radioactive iodide uptake of the gland and the protein-bound iodine of the blood plasma are elevated During treatment with any of the thiourea derivatives the gland becomes larger and softer and a bruit not previously heard may develop, after iodine therapy it becomes smaller and firmer and the bruit vanishes

Hyperplastic goiter may also be a manifestation of chronic iodide deficiency, a picture which is rarely encountered in this country today Systemic manifestations, if present, are those of low thyroid function, not of thyrotoxicosis The radiactive iodide uptake is high but the protein-bound blood iodine is normal or low

Nodular Goiter One or more fairly firm, discrete nodules appear in a normal or colloid gland They may be asymptomatic or cause thyrotoxicosis The latter, when present, can be due either to hyperfunction of the nodules or to hyperplastic changes occurring in the normal glandular tissue When hyperfunction of the nodule is responsible, the remainder of the gland is likely to appear atrophied In contrast to hyperplastic goiter, exophthalmos rarely accompanies the other signs of thyrotoxicosis

Carcinoma. The gland shows irregularly shaped, firm, stony hard, fixed enlargement, often with early metastatic swelling of the regional cervical nodes,



FIG 5 6 Non malignant, non-toxic, nodular goiter developing on right after hemithyroidectomy for similar growth on left

especially the Delphian nodes Signs of thyrotoxicosis are absent Any discrete nodule felt in the thyroid must be regarded as potentially malignant

Acute Thyroiditis. Tenderness and symmetrical enlargement of the gland, with redness and heat of the overlying skin are characteristic The Delphian nodes are usually palpable Fever, leukocytosis, and increased sedimentation rate are likely Thyrotoxicosis does not develop The disease is usually self limited; low thyroid function is occasionally the end result

Chronic Thyroiditis. There are two common forms the *Hashimoto* type, characterized chiefly by lymphoid infiltration of the struma, and the *Riedel* type, in which fibrosis is predominant. In the former there is diffuse, slightly irregular, sometimes tender enlargement, in the latter, diffuse, symmetrical, stony-hard enlargement with fixation to surrounding tissues In thyroiditis the gland is more likely to retain its normal contour than in carcinoma, is more firmly fixed and rarely accompanied by enlargement of regional lymphnodes except for those situated just off midline above the isthmus Long-standing cases, in contrast to cancer, may develop signs of hypothyroidism To differentiate between carcinoma and thyroiditis is rarely possible without biopsy.

CONGENITAL ABNORMALITIES

Cervical Rib. This anomaly, arising from the seventh cervical vertebra, and with a free end or attached to the manubrium or first rib, may be manifested just above the clavicle by angular fulness which occasionally pulsates where

the subclavian artery passes over the rib. The bone can sometimes be felt by careful palpation, but often is detected only by x-ray. Cervical rib may be asymptomatic but trouble may start at any time. The first symptoms are usually tingling and vasomotor changes in the fingers; later there are pain, usually of ulnar nerve distribution, diminished sensibility, and weakness and atrophy of some of the muscles of the hand and fingers. The vasomotor disturbance may proceed to trophic change or even gangrene of the fingertips. The radial pulse may be diminished or absent with the arm in its usual position or be weakened or obliterated by moving the arm into an extreme position, especially hyperextension Moving the head into an extreme position may aggravate symptoms and diminish pulse volume. Tight or hypertrophied scalenus anticus muscle (*Naffziger's syndrome*) or, less often, interference with circulation by some other muscular or ligamentous variant, can produce a comparable picture.

Branchial Cyst. A circumscribed collection of fluid in a persistent branchial cleft occurs along the anterior border of the sternomastoid muscle anywhere between the angle of the jaw and the sternal notch, most commonly beside the middle third. It presents as a rounded somewhat fluctuant mass which is tender only when secondarily infected. Variation in size may be observed as a result of its tendency to discharge intermittently into the pharynx or externally in the neck through a small cutaneous opening somewhere along the anterior border of the sternomastoid muscle (*branchial fistula*). The retained fluid is mucoid unless infected. Because of the thickness of its wall and its position beneath the platysma muscle, the cyst is not readily transilluminated The presence of a small dimple in the skin near the sternomastoid muscle, especially if associated with a history of periodic discharge, is of importance in differentiating this lesion from swelling due to some other cause, especially a suppurating lymphnode

Thyroglossal Cyst. Due to persistence of part of the fetal thyroglossal duct, this cyst occurs in midline of the neck between the hyoid bone and the suprasternal notch Its features are similar to those of branchial cyst



FIG 5 7 Circumscribed swelling beneath angle of jaw due to branchial cyst (Courtesy Dr David Weisberger)

Hygroma. This congenital abnormality of the lymphatic system presents a cystic swelling usually situated just above the clavicle at the base of the neck. It is single or lobulated, fluctuant, somewhat movable, contains either clear or serosanguineous fluid, and can be transilluminated. Its greater mobility and translucency differentiate it from branchial cyst.

Dermoid Cyst. Since the floor of the mouth is an occasional site, dermoid cyst should be considered when non-tender swelling in the submental region is encountered.

BLOOD VESSELS

Carotid Arteries

Normally, pulsation of the carotid arteries is not visible but can be felt on each side from the sternoclavicular junction to the angle of the jaw. To detect it, one presses the tips of 2–3 fingers obliquely inward and backward toward the spine at successive levels along the anterior border of the sternomastoid muscle. Pulsation to the level of the thyroid cartilage is from the common carotid, above this point, chiefly from its internal branch.

Visible pulsation. This is often observed in a normal person during strenuous exertion or an emotional upset, otherwise, it is most likely due to free aortic regurgitation, hypertension, thyrotoxicosis, or anemia. In a child it suggests coarctation of the aorta. Pronounced pulsation just above and to each side of the sternal notch can be caused by aortic aneurysm but in an older person is more apt to be due to arteriosclerosis of the carotids with or without hypertension.

Impaired pulsation. Diminished to absent pulsation of a carotid artery may be caused by external pressure as in intrathoracic tumor or aneurysm or dissecting aneurysm, or by intraluminal occlusion—thrombosis or embolism.

Palpation of the carotids is particularly important in a patient with hemiparesis, especially if transient attacks preceded a major episode or if vision had become impaired in the eye on the side opposite the affected limbs, this combination of symptoms is highly suggestive of carotid artery thrombosis. Support for the diagnosis can sometimes be obtained by detecting diminished to absent pulsation of the common or internal carotid on the pertinent side. If the common carotid is obstructed, pulsation will be impaired between the sternoclavicular junction and the angle of the jaw. If the common carotid is patent but the cervical segment of the internal is involved, absence of pulsation above the level of the thyroid cartilage is to be expected but one might be misled by feeling pulsation reflected from the adjacent external carotid. As a rule, this contingency can be excluded by palpating as high as possible in the neck, good pulsation here is almost certainly from the internal artery. In the equivocal case, some observers palpate with the forefinger directed outward and backward against the posterolateral wall of the tonsillar fossa: normally, pulsation of the internal carotid can be felt in this area; if it is not discernible, impaired blood flow in the vessel can be assumed. It must be remembered that thrombosis or embolization can occur in the petrous or intracranial segment of the artery without involvement of its cervical segment.

Jugular Veins

Dilatation and Abnormal Pulsation. Dilatation and abnormal pulsation, best observed in the external jugulars, are associated with congestive heart failure, less often with interference to return flow of blood, as in constrictive pericarditis, tricuspid valvular disease, mediastinal tumor, or thrombosis of the superior vena cava or a pulmonary artery (see Chap 8). It is sometimes possible to note, by inspection, abnormalities of the various waves of the jugular pulse, but such observations are not altogether reliable even when made by an experienced observer. When the study of these waves is regarded as important it should be performed by the graphic method Systolic pulsation of the deep jugular vein indicated by an outward movement of the sternomastoid muscle is highly suggestive of tricuspid valvular disease

Collapse. This is observed in peripheral circulatory failure due to diminution of circulating blood volume

THE TRACHEA

Palpation of the trachea is best carried out just above the suprasternal notch by grasping it between the thumb and first two fingers

Displacement. Lateral shift toward the *opposite* side is brought about by aortic aneurysm, mediastinal tumor, unilateral or asymmetrical enlargement of the thyroid, or a large amount of fluid or air in one pleural cavity Contraction of extensive adhesions, as in chronic fibroid pleuropulmonary disease, or increased negative pressure associated with obstructive atelectasis, may draw the trachea toward the *affected* side If tracheal displacement can be demonstrated, it is an important differential point in distinguishing between massive collapse of the lung and pneumonia or large pulmonary infarction, especially in a postoperative patient too ill to undergo detailed examination.

Forward displacement may result from abscess or tumor of the cervical spine, posterior goiter, or deep-seated mediastinal tumor This change is rarely appreciable on physical examination, but is readily seen in the lateral roentgenogram

Tracheal Tug. Standing behind the patient the observer grasps the trachea below the cricoid cartilage, gently but firmly between his thumb and fingers To bring the trachea forward the patient's head should be tilted backward If tug is present, a downward pull synchronous with cardiac systole will be felt Tug must not be confused with pulsation transmitted from the regional vessels, the latter, usually associated with free aortic regurgitation or hypertension, moves the fingers in and out, not up and down. Tracheal tug is most likely due to aortic aneurysm

6

THE SHOULDER GIRDLE AND UPPER EXTREMITIES

THE SHOULDER GIRDLE

Examination of joints is described elsewhere (see Chap. 7 and Chap 35).

Strain. In the scapulohumeral or acromioclavicular joint, strain or sprain occurs as a result of direct trauma, violent twisting motion or excessive, unaccustomed use Pain may limit all motion, *particularly abduction and rotation* Regional tenderness may be generalized, but is greatest over the joint affected

Fracture. Fracture occurring in a long bone produces swelling, limitation of motion, muscle spasm, local tenderness with increased pain on attempted motion, and perhaps crepitation In the shoulder region, fracture of the clavicle or surgical neck of the humerus is the most common

Dislocation. The indications are severe pain, swelling, gross deformity, marked limitation of motion, and muscle spasm There may be tenderness over the ligamentous attachments and evidence of fluid in the joint. Subcoracoid dis*location of the humerus is the most common; the classical signs are depression* lateral to the tip of the acromion and prominence of the humeral head beneath the coracoid. Dislocation of the acromioclavicular joint is indicated by upward displacement of the outer end of the clavicle, of the sternoclavicular joint by displacement, usually upward and forward, of its inner end

Injury to the Supraspinatus Tendon. Sustained from strain or direct trauma, injury to this tendon at its attachment to the humerus produces pain and limitation of motion The picture resembles that of acute bursitis except that *local* tenderness over the bursa is absent Complete rupture of the tendon is characterized, in addition, by inability to initiate abduction of the arm from the side.

Subdeltoid (Subacromial) Bursitis. Trauma, fatigue and chronic irritation, particularly occupational, are frequent etiologic factors, but often no cause can be discovered. Acute or chronic bursitis causes pain in the shoulder which may extend down the outer side of the arm and perhaps into the neck. Motions of the arm, especially abduction and external rotation, are restricted and painful The patient is likely to hold his arm inwardly rotated, close to his side, and with

the subacromial region. Atrophy of the deltoid muscle occurs fairly rapidly and

is remarkably persistent. Calcium deposits in the supraspinatus tendon, visible by x-ray, are often present

Brachial Neuritis. Usually secondary to cervical strain or disease of a cervical vertebra, this disorder is manifested by pain across the shoulder and down the arm The pain is aggravated by pressure over the cervical spine and by movement of the neck, particularly rotation, lateral bending or hyperextension; it is not affected by motion of the shoulder. Tenderness is found over the nerve trunks where they emerge from the spine and in the supraclavicular area, but not in the subacromial region, as in bursitis.

Shoulder-hand Syndrome. This term is applied to reflex neurovascular dystrophy affecting the shoulder and upper extremity and provoked by a number of different causes It is characterized by pain, tenderness, and limitation of motion of the shoulder, and usually aching, paresthesia, swelling, color changes, and limited mobility of the hand and fingers Stiffness, weakness, muscular atrophy, and flexion deformity of the hand and fingers with trophic changes of skin and subcutaneous tissue may develop in severe cases Among the causes described are those which are predominantly vascular, such as Raynaud's disease and thrombo-angiitis obliterans, predominantly neurologic, such as postherpetic herpes zoster and late hemiplegia, and others representing a combination of the two, such as cervical rib and scalenus anticus syndrome In some cases the cause cannot be determined The disturbance is occasionally seen following severe myocardial infarction, here the mechanism is not understood

Arthritis. Any form of arthritis may involve one or more joints of the shoulder girdle, producing varying degrees of swelling, redness, heat, limitation of motion, and deformity

Tuberculosis. The signs are swelling, increased heat, redness, local tenderness, limitation of motion, and, in advanced cases, possibly abscess formation and draining sinuses. The x-ray film shows a destructive process involving the head of the humerus and the glenoid cavity Aspiration of joint fluid for direct smear, culture and guinea pig inoculation may be necessary for diagnosis Tuberculosis rarely involves the sternoclavicular or acromioclavicular joint.

Anterior Poliomyelitis. Any or all muscles of the shoulder girdle may be involved, producing "flail" shoulder. The deltoid muscle is most frequently affected, in which event active abduction of the arm is impossible Normal sensation is maintained

Tumor. Sarcoma and other tumors occasionally involve the bones of the shoulder girdle (see Arms and Hands)

Neurotrophic Disorders. CHARCOT JOINT is rare in non-weight-bearing joints When present in the shoulder girdle, it is characterized by absence of pain, marked swelling, weakness, relaxation of ligaments and, sometimes, subluxation. It is caused by syphilis and, as a rule, there are also signs of tabes dorsalis X-ray will show a destructive process of the humeral head

SYRINGOMYELIA is more likely to cause joint involvement of an upper than of a lower extremity There are pain, swelling, destructive changes, relaxation, sometimes dislocation or fracture, but, in mild cases, little or no deformity.



FIG 6 1 Atrophy of muscles of shoulder girdle in progressive muscular atrophy.

Referred Pain. In addition to the local disturbances described above, pain in the shoulder is frequently referred from disorders elsewhere It is not likely, however, to be accompanied by local tenderness or limitation of shoulder motion

Referred pain can usually be traced to

1. Angina pectoris, coronary failure, or occasionally myocardial infarction
2. Disease of the cervical spine or spinal cord
3. Brachial neuritis
4. Disease of the mediastinum, such as tumor or aneurysm
5. Disease involving the diaphragm, such as diaphragmatic pleuritis or subphrenic abscess.
6. Cholelithiasis
7. Hiatus hernia

THE ARMS AND HANDS

Position and Deformity

Variations in the position of the arm and hand are observed in certain cerebral or peripheral nerve lesions and in association with diverse other disorders

Hemiplegic Arm. The joints of the elbow, wrist and hand are flexed and adducted.

Flail Arm. The arm hangs loosely at the side This deformity is common in cases of infantile paralysis, brachial plexus paralysis, obstetrical paralysis and other nerve injuries.

Wrist Drop. The muscles of the forearm, especially the extensors of the wrist, are weak The most likely cause is peripheral neuritis, as in plumbism, deficiency state, idiopathic polyneuropathy, or musculospiral paralysis from trauma



Fig 62



Fig 63

Fig 62 Bilateral brachial paralysis, obstetrical Internal rotation and adduction of arms (Courtesy Children's Hospital, Boston)

Fig 63 Obstetrical paralysis Atrophy of limb and flexor paralysis of hand



Fig 64 Wrist drop in lead poisoning (Courtesy Dr Joseph C Aub)



Fig. 6 5 Pronounced deformity of hands and fingers due to long-standing rheumatoid arthritis.

Hysterical State. The patient may hold one or more of his extremities in an abnormal or bizarre position. The symptoms and signs do not show the peripheral, segmental or cerebral localization seen in structural disease

Abnormal Position due to Pain. Effort to avoid or reduce local or referred pain may cause the patient to hold his arm and hand in an abnormal position

Bowing of Forearm. This occurs as a result of malunited fracture and in rickets and Paget's disease

Arthritic Hand. Any change from slight swelling and some limitation of motion, with or without redness, to striking deformity and loss of function, marked by ulnar deviation of the fingers, subluxation of the metacarpo-phalangeal and phalangeal joints, muscular atrophy, and ankylosis of the fingers in flexion or extension may be produced by any form of arthritis, including gout.

Contracture. Dupuytren's contracture, occurring almost always in adult males, results from proliferation and contraction of the palmar fascia. Gradually progressive, painless flexion of one or more fingers—most commonly the ring and little fingers—of one or both hands is brought about by the contraction. The proliferated fascia follows the courses of the flexor tendons of the affected fingers, it can be felt in the palm as hard, tense bands fixed to the overlying skin

Volkmann's contracture, due to disturbance of the blood supply from injury in the region of the elbow joint, or sometimes from an overtight surgical dressing, causes fibrosis and contracture of the muscles The most likely deformity is flexion of the wrist and fingers (*claw hand*). Injury to the ulnar or median nerve may cause a similar type of deformity affecting the fingers, which receive their motor innervation from the involved nerve. In progressive muscular atrophy, *syringomyelia and some cases of leprosy, comparable contractures* and deformities occur

Enlargement. A thick, stubby hand (*spade hand*) is sometimes seen in normal persons. In myxedema, thickening and coarsening of the skin and soft tissues *without* bony change will produce spade hand In acromegaly, the hand becomes enlarged as a result of overgrowth of bone and soft tissues.



FIG 6 6 Dupuytren's contracture, fourth and fifth digits

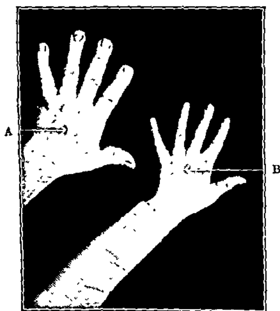


FIG 6 7 A Acromegalic hand B Normal hand

Redness of Palms. In chronic uncompensated liver disease, severe malnutrition and hypoproteinemia from any cause, the thenar and hypothenar eminences of the palms often show a characteristic mottled, red color (*liver palms*)

Clubbed Fingers. The nails are convex in both anteroposterior and lateral planes; proximally they are elevated from the matrix and movable on pressure

(*floating nails*). In all but the earliest cases, the fingertips show bulbous enlargement. Soft tissue swelling and often fraying of the ungual tufts can be seen by x-ray. Similar but less readily apparent changes may be observed on the toes. Clubbed digits are sometimes seen in normal persons without disease; the *tendency may be hereditary. They are also the most common form of pulmonary* osteoarthropathy—a peculiar, hypertrophic change involving all the tissues of the extremities, usually localized to the terminal phalanges, but sometimes seen in a more generalized form (see Chap. 35) This phenomenon may appear quite rapidly and will partially or totally disappear if the cause is alleviated or removed

The most common causes of clubbed fingers and toes are:

1 High altitude living

2 Cyanotic types of congenital heart disease.

3 Subacute bacterial endocarditis and, rarely, rheumatic heart disease.

4 Suppurative pleuropulmonary lesions, particularly lung abscess and bronchiectasis

5 Primary and, rarely, metastatic bronchopulmonary malignant disease

6 Chronic liver disease, long-standing intra-abdominal tumor, ulcerative colitis and other chronic intestinal disturbances, especially when malnutrition is pronounced These causes are not as common as the others

Unilateral clubbing occasionally results from delay of venous return from one arm, as in aneurysm or intrathoracic tumor

Clubbed fingers must not be confused with the shortening and broadening of the tips sometimes seen in hyperparathyroidism Here nail changes are absent and the x-ray may show marked absorptive changes of the terminal bones



FIG. 6 8 Clubbed fingers in a patient with long-standing bronchiectasis.

Atrophy. The girth of the part is diminished; when atrophy occurs before growth is attained, longitudinal development is also retarded. Atrophy results from disease or injury of the anterior horn cells or peripheral nerves or from disturbance of blood supply, its degree obviously depending on the extent of underlying damage It is also seen in the various forms of joint disease, being especially pronounced in rheumatoid arthritis Atrophy may result from disuse from any cause and in association with subdeltoid bursitis. It is a feature of the rare disease, primary pseudohypertrophic muscular dystrophy.

Injury

Strain. Trauma or abnormal use of an elbow, wrist or finger will result in pain increased by motion, swelling, limitation of motion and local tenderness.

Fracture. The common sites are:

1 Humerus—surgical neck, middle or lower third

2. Elbow—supracondylar, head of radius, olecranon process.

3 Forearm—upper, middle or lower third of one or both bones, with slight or marked separation of fragments

In children, fracture is often incomplete and may be subperiosteal (*greenstick*). Fracture of the lower end of the radius (*Colles's fracture*), by far the most common break of the upper extremity, occurs as a result of forced dorsiflexion of the hand on the forearm, such as occurs with a fall on the outstretched hand. The lower fragment is forced upward and backward upon the shaft of the radius, giving a characteristic *silverfork* deformity Fracture of a carpal bone, particularly the scaphoid, is common, it is often misdiagnosed sprain It cannot be positively diagnosed without x-ray

Dislocation. The most common dislocations are:

1 Posterior dislocation of the ulna on the humerus.

2 Dislocation of the semilunar bone

3 Posterior dislocation of a phalangeal bone on its proximal neighbor

Abnormalities of Motion

Manner of Shaking Hands. This provides a general impression of the patient's temperament The nervous, cramped, half-opened hand which never really grasps and is quickly withdrawn, the firm hearty grasp, and the limp handshake all give clues to the personality pattern.

Weakness or Paralysis. The patient's ability to flex, extend, abduct, adduct, and rotate the arms, hands, and fingers—freely and against resistance—should be tested Weakness or paralysis of part or all of one upper extremity occurs in a variety of neurologic diseases, such as cerebral thrombosis, hemorrhage or tumor, multiple sclerosis, paralysis agitans, anterior poliomyelitis, and in such disorders as cervical rib, subacromial bursitis and Volkmann's paralysis Parts or all of both may be affected in various forms of peripheral neuropathy, multiple sclerosis, paralysis agitans, anterior poliomyelitis, progressive muscular atrophy, and other neurologic disturbances Paresis must not be confused with inability to move a part because of pain

Carphologia. This term applies to the picking and fumbling at the bedclothes seen in severe illness, often with muttering delirium. It often accompanies typhoid fever, pneumonia, the toxic delirium resulting from alcohol, drugs or other toxemic states. Generally speaking, it is a grave sign.

Subsultus Tendinum. This is involuntary twitching and jerking of the tendons of the wrist and dorsum of the hands, usually associated with tremor and carphologia.

Tremor. Tremor may be coarse or fine. Test for fine tremor is best performed by having the patient hold the arms at right angles to the trunk with hands extended, palms down, and fingers spread. When not apparent by this method, it can sometimes be detected by having the patient hold a glass of water or by placing a piece of paper on the dorsum of his outstretched hand and fingers.

The common causes of tremor are:

1. Nervousness, cold, old age, and prolonged, unaccustomed use of hands or arms.
2. Fever and toxemia.
3. Alcohol or drugs in excess, or following withdrawal.
4. Thyrotoxicosis.
5. Parkinsonian syndrome.
6. Multiple sclerosis.
7. Cerebellar disease.
8. General paresis.
9. Hysteria and disturbed emotional states.

In most of these, tremor is intensified on voluntary movement; in Parkinsonian syndrome it is likely to be less pronounced.

Often in impaired sensorium due to hepatic failure, less frequently when it is due to other cause, one can demonstrate peculiar flapping movements of the hands and fingers, sometimes even of the arms (liver flap).[1] These are best brought out by having the patient hold the two extremities outstretched and pronated, with the fingers apart. The movements consist of jerky, flexion-extension with a tendency for the fingers also to deviate laterally. They occur in short cycles of several per second with varying intervals, usually 1–2 seconds between. In addition, a fine tremor of the digits may be seen. (The lower extremities may show similar movements, they can be demonstrated on either side by having the patient, while recumbent, elevate his leg and dorsiflex his foot).

Fasciculation. Spontaneous, irregular twitching of muscle fibers is seen in progressive muscular atrophy, syringomyelia, peroneal muscular atrophy (*Charcot-Marie-Tooth*), and some cases of polyneuropathy. It is common in uremia. Fasciculation must be distinguished from simple shivering or the shaking that is characteristic of nervousness or fatigue.

Choreiform Movements. These irregular, spasmodic movements and twitchings of the fingers, hands, and arms can be described as pseudo-purposive, since

[1] Adams, R. D., and Foley, J. M. Proceedings of the Association for Research in Nervous and Mental Disease 32, 1953.

they give the impression of being intentional but are seldom carried through to their logical ends and are actually involuntary. They make the patient appear restless. These movements occur in Sydenham's chorea, Huntington's chorea, occasionally during and following pregnancy (thought by most observers to be a recrudescence of previous Sydenham's chorea), and in some cases of cerebral arteriosclerotic degeneration Occasionally, one-sided involvement occurs (*hemichorea*); in a number of these cases, autopsy has revealed hemorrhage in the subthalmic region

Athetosis The patient shows rather slow, almost constant involuntary movements of a hand or wrist or sometimes of the whole extremity Combinations of flexion, extension, rotation and lateral motion produce a variety of bizarre, serpentine, clutching, spreading or bending movements of the involved parts Athetosis is due to organic cerebral disease, occurring most likely as a late sequel of hemiplegia or encephalitis It is intensified by voluntary movement and cannot be controlled by voluntary effort. It does not occur in a totally paralyzed part. Bilateral athetosis, usually congenital but sometimes not evident until childhood, is seen most often in spastic paraplegia; it may be accompanied by grimacing of the face and difficulty in articulation

Tetany. Hyperexcitability of the nervous system causes tonic spasm of the muscles. It is of two types (1) *hypocalcemic*, due to parathyroid insufficiency, inadequate calcium intake or absorption, renal insufficiency with phosphate retention, or lack of vitamin D, and (2) *alkalotic*, caused by excessive alkali intake, hyperventilation or persistent vomiting with loss of chloride. The muscle spasm may be continuous for several hours, sometimes for days, or it may be intermittent, lasting only a few minutes and appearing at intervals of hours or days It may be general or local and shift from one part to another Usually it involves the extremities (*carpopedal spasm*), face and larynx The hand is flexed at the wrist The fingers are flexed at the metacarpophalangeal joints; distally they are stiff, extended and separated The thumb is flexed and adducted across the palm If not immediately apparent, carpal spasm can be brought out by applying sufficient pressure around the upper arm to obliterate the radial pulse (*Trousseau's sign*) If obtainable, this sign is conclusive evidence of tetany



FIG 6 9 Position of hand in tetany (Courtesy Dr Joseph C Aub)

In doubtful cases, hyperexcitability may be demonstrated by the galvanic current (*Erb's sign*).

Edema

In the arm, as elsewhere, subcutaneous edema causes swelling, which can be distinguished from obesity and other enlargement by the fact that a dent made by pressing the part will not disappear promptly following withdrawal of pressure Edema, if due to general rather than local disease, varies with changes in the patient's position because of the tendency of fluid to accumulate in the dependent parts

The common causes of edema of the arm are:

1 Inflammation due to local trauma, infection or an overlying skin disorder.

2. Thrombophlebitis of one of the larger veins

3 Axillary scar tissue or enlarged lymphnodes, especially when malignant.

4. Intrathoracic disease causing delay in return flow of blood or lymph, such as tumor or aneurysm.

5. *Certain neurologic lesions, such as hemiplegia and peripheral neuropathy* Here the swelling, which is probably of trophic origin, is rarely pronounced.

The Skin

The character and distribution of skin lesions on the upper extremity are important in the differentiation of a number of dermatologic and systemic disorders

Measles. The eruption is blotchy and favors the extensor aspects.

Rubella. The eruption is blotchy but finer and shows no predilection for any particular site

Scarlet Fever. The punctiform rash is more accentuated on the flexor aspects and usually best seen in the bend of the elbow and on the inner aspect of the upper arm Application of a tourniquet to the upper arm will sometimes aid in establishing the diagnosis by bringing out, within a few minutes, small petechial hemorrhages in the fold of the elbow. This test is also used in diagnosing purpura

Syphilis. Cutaneous lesions of secondary syphilis similar to those on the trunk are usually present on the arms, the palms show yellowish-brown or ham-colored macules or patches of irregular, roundish or circinate shape. In young infants glossiness of the palms and soles is an important diagnostic feature of congenital syphilis; occasionally one sees a palmar eruption similar to the secondary rash of acquired syphilis

Pellagra. The skin of the hands, especially of their dorsal surfaces, is at first red and shiny but later shows brownish pigmentation. It may be confused with sunburn. Cracking, vesiculation and desquamation often develop. Atrophy may be the end result. Especially characteristic is the symmetrical cuff-like distribution over the hands, wrists and perhaps the forearms, with a clear-cut line of demarcation between normal and abnormal skin The forehead, neck and feet may be similarly involved.

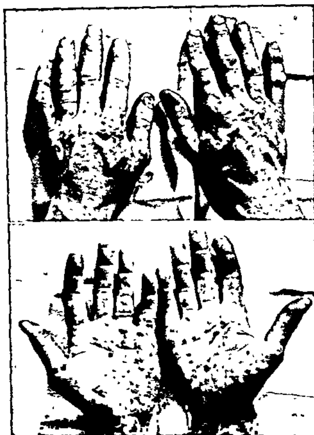


FIG 6 10 Arsenical keratosis

Vascular Disease. In Raynaud's disease, endarteritis obliterans, disseminated lupus erythematosus, and allied disorders such as scleroderma and dermatomyositis, the skin of the fingers may be shiny, atrophic, or, sometimes, thickened, and show variations in color such as cyanosis, redness, and pallor

Rheumatoid Arthritis. An atrophic, shiny appearance of the skin of the hands is frequently seen, pigmentation is common.

Arsenical Keratosis Chiefly on the palms and extending along the sides and between the fingers, the skin is irregularly thickened and shows small hard callosities Areas of erythema may surround the horny thickenings

Scabies. The lesions are seen in the webs of the fingers, on the palms and flexor aspects of the wrists Usually there are typical burrows, vesiculopustular lesions and excoriated papules Impetiginous lesions from secondary infection may be present

Tularemia. Since the primary lesion is often found on a hand or finger, any indolent ulcer with raised edges and a punched-out appearance, especially if accompanied by axillary lymphnoditis and constitutional symptoms, should suggest the possibility of tularemia (see Chap 3).

Dermatologic Disorders. Cutaneous diseases commonly appearing on the upper extremities are erythema multiforme, impetigo contagiosa, pityriasis rosea, lichen planus, dermatitis venenata, contact or occupational dermatoses, epidermophytosis, and warts

Temperature and Moisture of Hands

Cold Moist Hands. Most commonly associated with emotional strain or instability, cold, moist hands are also found in local vascular or vasomotor disturbances such as Raynaud's disease and other circulatory or neurotrophic disorders They are common in rheumatoid arthritis, but if the joints of the hands or fingers are acutely involved, the hands may be moist but warm. They are also found along with generalized coldness and moisture of the skin in peripheral circulatory failure. There is no foundation for the superstition that they mean heart disease

Cold Dry Hands. The hands (and feet) are often cold and dry in chronic wasting disease and sometimes in the circulatory disturbances mentioned above.

Warm Moist Hands. Common in normal children, warm moist hands in the adult suggest thyrotoxicosis, and make a search for other manifestations of the disease imperative If the hands are *sweaty but cold,* they are, in the opinion of many competent observers, strong evidence *against* thyrotoxicosis

Hot Dry Hands. The most likely cause is fever

The Joints

The several stages and types of rheumatic fever, arthritis, gout, pyogenic infection, neurotrophic disorder, and other disturbances affecting the joints, produce varying degrees of pain, swelling, deformity and limitation of motion The swelling may be confined to the periarticular tissues or be partly the result of



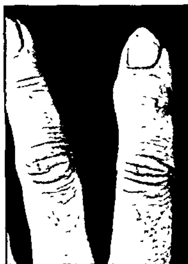


FIG 6 12 Cystic swelling overlying terminal joint of index finger Such a change is often seen prior to development of a Heberden's node

changes in the synovial, cartilaginous or bony structures Fluid within the joint capsule is indicated by considerable swelling with fluctuation on palpation In acute rheumatic fever one finds redness, swelling, increased surface temperature and limitation of motion Similar findings may be seen in active rheumatoid arthritis; spindle-like swellings around the proximal joints of the fingers are also common. In acute gout the joint is greatly swollen and tender, with the overlying skin red and shiny; regional veins may be distended. In chronic gout the picture resembles that of a chronic arthritic joint Tophi are usually present (see below) *Heberden's nodes,* occurring in hypertrophic arthritis, are bony



FIG 6 13 Degenerative joint disease in a woman age 50 Terminal phalangeal joints show deformity and well defined Heberden's nodes



Fig 6 14 Chronic gouty arthritis of 15 years' duration in a man age 50 Deformity and swelling of fingers with striking tophus formation on left fourth and right fifth

outgrowths at the terminal and mid-phalangeal joints, usually accompanied by some deformity These must be distinguished from tophi. Disease of a joint is often accompanied by protective spasm or atrophy of the associated muscles

Localized Swelling

Trauma, pyogenic infection of the skin and subcutaneous tissue, and various forms of joint disease are among the most common causes of localized swelling of an upper extremity Others which merit description are:

Tenosynovitis. Common on the wrist or forearm, tenosynovitis is inflammation of the tendon sheaths, acute or chronic, suppurative or nonsuppurative It is often traumatic, resulting from constant repetition of some motion; it may be tuberculous Pain, sometimes with crepitation on moving the part, moderate swelling and perhaps redness over the affected tendons, are characteristic

Ganglion. Popularly known as *weeping sinew*, ganglion is a firm, fluctuant, usually painless swelling, most often found on the dorsum of the wrist or hand It represents outpocketing of a tendon sheath or of the synovial lining of the wrist or a carpal joint

Rheumatic Nodules. Subcutaneous nodules often appear during the active stage of severe rheumatic infection, and in rheumatoid arthritis These are small, discrete, non-tender swellings which, on an upper extremity appear usually over *a tendon or the bony prominence of the elbow, wrist or a finger. They vary in* size from a few millimeters to larger conglomerate masses one or more centimeters in diameter. A nodule is best seen with the joint flexed, so that the skin is stretched and whitish over the prominence When overlying a tendon it is *usually attached* to the sheath but not to the skin. It is distinguished from a bony prominence by being movable and usually bilateral.

Gouty Tophi. These are hard, non-tender, white or yellowish, subcutaneous nodules which, in their early stages, may closely resemble rheumatic nodules and be distinguishable from them only by microscopic examination Like rheumatic nodules they tend to grow to a diameter of one or more centimeters They



Fig 6 15. Subcutaneous nodules distal to olecranon process in a man age 51 with rheumatoid arthritis of 3 years' duration

may become inflamed and ulcerate. In addition to the helix of the ear (see Chap 4) Favored sites are the dorsum of the hands and feet, the phalangeal joints and olecranon and prepatellar regions

Nodules of Polyarteritis Nodosa. In polyarteritis nodosa, careful palpation may reveal small (1–2 mm diameter), firm nodules in the subcutaneous tissues In suspicious cases, one searches for them not only along the courses of the larger vessels but throughout the extremities (see Chap. 35)

Enlarged Epithrochlear Lymphnode. Situated in the fossa above and behind the mesial condyle of the humerus, this node can best be found by palpating the region with the patient's arm relaxed and the forearm in partial flexion. Normally it is barely palpable if at all, so that an easily felt node is apt to have pathologic significance Enlargement is most likely due to local inflammation of the hand or forearm, or one of the diseases causing generalized lymphnodopathy

Swelling of Fingertip. The sudden appearance in a fingertip or toe of a small area of swelling, induration and redness which regresses within a few days is often encountered in subacute bacterial endocarditis In a suspected case, this finding has considerable diagnostic importance. Such lesions are tender, seem deep-seated and tend to appear in crops affecting several digits at about the same time They are thought to be due to small emboli

Osteomyelitis. Most often seen in children and young adults, this suppurative disease tends to attack the end of a bone, although any part may be involved Local swelling in the hand or arm, tenderness, and, later, redness and fluctuation, are the characteristic signs. The tenderness appears to be of bone, that is, it is deep-seated and can be elicited only after pressure with the examining fingers has been exerted steadily for a few seconds or longer This feature helps to distinguish osteomyelitis from superficial abscess Constitutional manifestations of infection, often severe, are present In the early stages, x-ray reveals *no evi-*



FIG 6 16 Tophi overlying terminal phalangeal joint of index finger in a man age 40 with gouty arthritis of 6 years' duration Note similarity to Heberden's nodes In a doubtful case aspiration of a nodule might be necessary for diagnosis





A B

FIG 6 17 A Tophus of olecranon bursa in a patient age 60 with gouty arthritis of 25 years' duration On palpation the swelling appeared full of gritty material
B X-ray shows that swelling is not due to calcium deposits or bony change

dence of disease but within approximately three weeks of onset, thickening of the periosteum is seen, followed by bone destruction and sequestration Long-standing cases show multiple sinuses and thickening of bone Osteomyelitis may be simulated by Ewing's tumor.

Tuberculosis. Tuberculosis of an upper extremity is most likely to involve the wrist, less often the elbow, shoulder, or a finger. Beginning in the epiphysis or synovial membrane, the lesion progresses slowly, causing swelling, heat, local tenderness, limitation of motion, protective muscle spasm, probably deformity, and possibly abscess formation. Long-standing cases may show draining sinuses leading from necrosed bone

Syphilis. The long bones are variously affected Most common is syphilitic periosteitis, characterized by thickening of the cortex and induration which feels like the callus of a healing fracture Pain, especially at night, and moderate tenderness are the rule. Congenital syphilis sometimes produces painless swelling around the joint, often with fluid (*Clutton's joint*, see Chap 35) Although the knee is the most common site, an elbow or shoulder is occasionally affected. A history of other more characteristic lesions of syphilis, x-ray, and serologic tests may be necessary to establish diagnosis.

Dactylitis. One or more phalanges, usually the proximal, show fullness or spindle-shaped swelling, some redness and moderate tenderness resulting from pyogenic infection, tuberculosis, syphilis or other granulomatous disease Periosteitis, central bone destruction, abscess and perhaps sinus formation develop. Diagnosis can be established only by x-ray and other means of investigation In sarcoidosis suppuration rarely occurs, x-ray shows sharply defined areas of rarefaction without periosteal or joint involvement, except perhaps for peripheral pressure erosion.

Benign Tumor of Soft Parts. KERATOSIS has already been described (see Chap 3).

LIPOMA of the upper extremity, usually occurring in the upper arm or shoulder, is recognized by history of long duration, slow growth, superficial position exterior to the muscles, and lobulated feel It seems circumscribed and does not give the impression of being invasive Rarely the mass is situated deeply and discovered only on x-ray

NEUROFIBROMA, a small circumscribed tumor felt in the skin, is frequently multiple and occurs along the nerve trunks

FIBROSARCOMA is a term loosely applied to a non-metastasizing cellular fibrous tumor, as well as to the highly malignant anaplastic tumor arising from the intramuscular fibrous septum

GIANT CELL TUMOR of the tendon sheath is usually seen on the finger as a rounded swelling, most often on the flexor tendon.

GLOMUS TUMOR, a non-malignant tumor arising in the digital glomus system, occurs as a pea-sized, firm, exquisitely tender nodule, usually beneath a nailbed

hand in an elderly person. It has the same characteristics as similar lesions elsewhere but tends to metastasize early

SARCOMA arising from fascial, synovial, lipoid or neural tissue is movable, often slightly tender, circumscribed and although often giving the impression of being non-invasive, it is usually highly malignant. It appears in any part of the arm

MALIGNANT MELANOMA arises in a pigmented mole (see Chap 3) A particularly malignant form begins beneath the fingernail At first it shows slight swelling, tenderness, and perhaps mild suppuration, and is often mistaken for paronychia from which it can be distinguished only by its black color. This tumor metastasizes with amazing rapidity.

NEURO-EPITHELIOMA is a rare form of malignant tumor If found on the arm it is usually situated around the elbow joint

KAPOSI TUMOR appears as a small angioma of the skin, most often on a hand or foot It is slow-growing, apt to be multiple, and may ulcerate in the late stages. It is associated with similar lesions in the gastro-intestinal tract, the patient eventually dies from generalized malignant disease.

Benign Tumor of Bone. Single tumor of a small bone is most likely chondroma or giant cell tumor.

SOLITARY CENTRAL CHONDROMA, usually found on a finger, may cause slight, local, painless swelling or be discovered by x-ray. Pathologic fracture may be the first indication

GIANT CELL TUMOR occurs at the epiphyseal end of a long bone, most commonly the radius or ulna At the upper end of the humerus, it may be confused with bone cyst A malignant variant is described below

MULTIPLE OSTEOCHONDROMAS of the upper extremity are seen on the bones of the hand or about the epiphyses of the arm, and are usually associated with similar lesions involving bones elsewhere This disease, also known as *multiple chondralexostosis*, is frequently familial The growths are hard, circumscribed, obviously attached to bone and often overlain by bursae; they may become malignant

Malignant Tumor of Bone. The earliest manifestation of malignant tumor of bone is either pain or the appearance of a palpable or occasionally visible mass, noted by the patient himself. The pain is characteristically described as *a severe bone ache which is apt to be worse at night, it is not relieved by immobilization* Any malignant tumor, if sufficiently advanced, will show redness, increased surface temperature, and dilated superficial veins, but by the time these indications have appeared, it is usually too late to effect a cure. For this reason, *any complaint of constant ache or pain in the arm not readily explained by some simple cause is indication for immediate roentgenologic examination.* All malignant tumors of the bone metastasize to the lung early, in fact, symptoms referable to the lung often appear before those of the primary lesion X-ray of the bone may be sufficiently characteristic to establish diagnosis; if not, biopsy must be performed

OSTEOGENIC SARCOMA (OSTEOSARCOMA) of the upper extremity is most com-

monly found at the upper end of the humerus. It is rare in the metacarpals and phalanges Bone sarcoma is hard, slightly tender and obviously originates in bone. Pain, unrelieved by rest or immobilization, is the first indication; later, swelling appears.

EWING'S SARCOMA, often mistaken for osteomyelitis, arises in the shaft of a long bone, especially the femur or fibula, but may be encountered in any of the three bones of the arm. Pain and a palpable mass, accompanied by local heat and redness and perhaps slight elevation of temperature and leukocyte count, are characteristic. Diagnosis is established by roentgenologic examination.

METASTATIC MALIGNANT DISEASE may appear in any bone. The most common sites are the spine, pelvis, skull, thoracic cage and region of the knee. Symptoms are similar to those of primary malignant disease Spontaneous fracture is sometimes the first sign The indications of a metastatic lesion may precede those of the primary lesion, in such a case, one looks for the primary focus first in the breasts, kidneys, prostate, lungs and thyroid.

MULTIPLE MYELOMA, which is most likely to involve the skull, spine, thoracic cage and pelvis, but rarely the long bones, simulates metastatic malignant disease If, by x-ray, the bone lesions appear osteoblastic, myeloma is ruled out; if they are osteoclastic one cannot distinguish roentgenologically between myeloma and metastatic malignant disease

THE ARTERIES

Palpation of the pulse waves and walls of the brachial, radial and ulnar arteries, as well as those of the corresponding vessels of the lower extremities, is essential in the diagnosis of arteriosclerosis, Raynaud's disease, Buerger's disease, and peripheral embolism Other important factors are: determination of surface temperature, nutritional state of the skin and subcutaneous tissue, and variations in color and temperature in response to positional changes of the limb, external temperature changes, and emotional stimuli The pulse is described in chapter 9, the various forms of arterial disease as they affect the lower extremities, in chapter 34

Raynaud's Disease. A disturbance of the autonomic nervous system predominant in young women, this is manifested by vasospasm of the arteries, arterioles and capillaries of the distal portions of the extremities, the larger arteries and their branches higher up remain normal It is more common in the hands than in the feet The extremities are subject to repeated episodes of local ischemia, usually bilateral and symmetrical, and precipitated by cold or emotional tension One or more fingers are always involved, the palms and dorsums occasionally, the thumbs, rarely At the start, the affected parts become cold, numb or definitely painful, cyanotic, pallid or dead-white, and cold to the touch A sharp line of demarcation is evident between the ischemic and non-ischemic zones This status is maintained until the precipitating factor is alleviated, whereupon a hyperemic reaction, indicated by burning and redness, supervenes, followed by gradual return to normal. Between attacks the fingers and hands are likely to be cold and moist and, in some cases, a cyanotic tint is maintained



Fig 6 18 Raynaud's disease Ischemia of fingers distally Thumb not involved



Fig 6 19 Raynaud's disease, advanced Scleroderma and ulceration of fingertips Cyanosis of fingers and palms present but lost in reproduction

The changes indicated can be reproduced by immersing the hand in cold water *It is not uncommon to encounter a mild and non-progressive form in which* one or more fingers become cold and pallid on exposure and promptly return to normal when warmed, with nothing more than recurrent temporary annoyance. On the other hand, if the case is of long duration, attacks occur frequently or vasospasm becomes virtually permanent, one may find trophic changes such as

scleroderma, impaired nail growth, ulceration, and eventually gangrene of the terminal phalanges.

Similar variations can also occur in the presence of obliterative arterial disease or scleroderma, and in persons habitually using vibrating tools (Raynaud's phenomena).

Arteriosclerosis. In the early stages due to fibrous thickening, the artery has a smooth surface, is firmer than normal but not incompressible. As a rule, it can be rotated under one's finger. Tortuosity appears later, and finally, calcification. The latter, readily demonstrable by x-ray, produces beading, felt as a series of rough elevations or transverse ridges along the course of the vessel, or is diffusely distributed, creating a hard, incompressible tube (*pipestem artery*). Even when the process in an arm has proceeded to such a point that the artery is non-compressible and the pulse virtually impalpable, subjective symptoms are rare.

Thrombo-angiitis Obliterans. Buerger's disease is regarded as a generalized proliferative inflammatory process of both the arteries and veins. In contrast to arteriosclerosis, it begins in early or mid-life and is rarely encountered in women. Trouble predominates in the lower extremities but is by no means uncommon in the upper. Rare instances of involvement of the cerebral, coronary and mesenteric vessels have been reported. The process may be slowly or rapidly progressive and is often segmental. Changes secondary to ischemia are dependent on the number and size of the arteries involved; because of the facility with which collateral circulation will develop in unaffected vessels, the patient may get along for years without serious trouble. When circulation in an arm does become impaired, one usually finds diminution of arterial pulsation at the wrist and higher. The fingers are usually pale or cyanotic, especially at their tips. Symptoms may be absent or the patient will experience intermittent attacks of ischemia simulating Raynaud's disease. Ulceration and gangrene with perhaps ultimate loss of one or more digits are likely in the advanced case. Recurrent attacks of transient thrombophlebitis affecting short segments of the superficial veins of an extremity are common (*migratory thrombophlebitis*); these are marked by small, red, firm, tender areas obviously over veins but not confined to any particular vein. When they occur in association with evidence of arterial impairment, they are regarded as pathognomonic of thrombo-angiitis obliterans. Calcium deposits in the arteries are rare unless there is an accompanying arteriosclerotic process.

Peripheral Embolism. In an upper extremity, occlusion of an artery by a dislodged thrombus is most likely to occur where a major branch leaves the axillary artery, occasionally at a bifurcation lower down. Pain beginning shortly after the episode usually occurs first, followed by tingling, diminution of sensation, pallor, coldness, and impairment of muscular activity or sometimes actual paralysis of the part. These changes never extend as high up as the point of obstruction. Arterial pulsation is absent distally from the occlusion.

In arteriosclerosis, sudden occlusion from thrombosis can create a similar picture, but this is not common. To differentiate between the two is clinically impossible although embolus can be assumed if there is an underlying dis-

turbance predisposing to peripheral embolic phenomena, such as mitral stenosis with auricular fibrillation, recent myocardial infarction, or subacute bacterial endocarditis

The Veins

Distention. The degree of distention of the arm veins provides a rough index of venous pressure. Normally, the veins appear full or distended if the arm is below the level of the auricle, flat when above it.

If not due to some local lesion, distention of the veins when the arm is held above auricular level suggests cardiac or other intrathoracic disease (see Chap. 8)

Collapse. In peripheral circulatory failure, the veins of the extremities lack their normal fullness because of diminution of circulating blood volume. If a vein is compressed it may not even fill beyond the point of pressure

Thrombophlebitis. In an *axillary* or *brachial* vein, where this sometimes occurs, it is usually secondary to an infectious process, or to occupational trauma resulting form repeated motion of the arm, especialy one involving elevation Pronounced swelling and perhaps cyanosis are present distally In the earlier stages, one finds tenderness along the vein; later, it has a cord-like feel The collateral superficial veins are visibly dilated on the upper arm and adjacent chest wall Febrile reaction is common when the trouble is due to an infectious process, rare in the traumatic cases Thrombophlebitis of the *superficial* veins is common because of the frequency with which it follows intravenous therapy Local redness and tenderness are found along the involved vessels which are readily palpable, peripheral edema is absent The end result is a cord-like vessel Recurrent, migrating attacks of superficial thrombophlebitis are common in thromboangiitis obliterans

The Lymph Vessels

The superficial lymph vessels are not normally visible. In cases of local streptococcus or, less often, other pyogenic infection of an extremity, they are seen as red streaks extending along the courses of the superficial veins upward from the site of infection Regional lymphnodes are usually enlarged and tender In filariasis, recurrent attacks of acute lymphangiitis are common When an extremity is involved, the affected lymphatic vessels appear as red streaks and feel cord-like. Overlying skin is diffusely reddened and the limb is often edematous Regional lymphnodes are swollen and tender In contrast to the more common form of lymphangiitis, the process is retrograde, tending *to spread distally, not proximally.*

The Nails

Absence of Nails. This rare congenital anomaly is usually associated with absence or scanty growth of hair or with congenital ichthyosis. It may occur in families. Shedding of the nails may occur after local trauma or in disturbances causing severe dystrophy.

Concave Nails. So-called *spoon nails* are due to abnormal growth of matrix causing detachment and outward bending of the free edges Rarely this variant is congenital. Otherwise it occurs as part of a dystrophic process. In idiopathic microcytic anemia and Plummer-Vinson syndrome, the nails are often flattened or slightly concave and may be ribbed.

Convex Nails. These are most often one of the manifestations of clubbed fingers

Brittle Nails. Nails that break or split easily may occur without known cause. Brittleness is also one of the early features of dystrophy.

Leukonychia. In cases of imperfect nail growth, white spots, representing intercellular, air-filled spaces left by dead cells, appear singly or multiply on the nail following mild trauma.

Paronychia. ACUTE suppuration of the soft tissue about the nail results from infection secondary to hangnail or trauma

CHRONIC suppuration may result from low-grade bacterial or yeast infection; the latter is common in housewives and others who handle fruits and vegetables. Indolent sores around the nail suggest lowered general resistance; in children especially, tuberculosis or syphilis should be excluded

Dystrophy. The nails are lusterless, opaque, discolored or leukonychitic, furrowed, pitted or ridged and brittle They may become atrophic or hypertrophic and, in severe cases, detached Common causes are

1 Physical or chemical irritation, as in chemical workers, dishwashers and laundresses.

2. Local infection, as in chronic paronychia, dermatophytosis, favus, and other fungus diseases.

3 Cutaneous diseases, such as psoriasis, eczema, alopecia areata, contact dermatitis, and exfoliative dermatitis

4 Constitutional diseases, such as endocrine disorders, deficiency states, rheumatoid arthritis, syphilis, leprosy and severe or protracted febrile states Following severe, acute illness and, sometimes, pregnancy, a transverse ridge or groove may form at the proximal part of the nail and progress distally as the nail grows; it usually requires five to six months to reach the free edge. In syphilis a primary sore occasionally occurs in the peri-ungual tissues, most likely in a dentist, physician or nurse In the early stages of late syphilis, nail dystrophy may occur, it has no distinctive appearance.

5. Trophic disorders, such as peripheral nerve injury, peripheral vascular disease, and scleroderma

Splinter Hemorrhages. Small, linear, petechial hemorrhages are often seen beneath the nails in subacute bacterial endocarditis. In a suspected case, they assume diagnostic importance and should be looked for from day to day This phenomenon is also encountered in some cases of acute trichinosis, if hemorrhages occur they are likely to be more numerous than in endocarditis.

Discoloration Chemical workers often have color changes in the nails depending on the color of the responsible agent Topical applications such as

potassium permanganate, gentian violet, bichloride of mercury and others also cause characteristic discoloration. A grayish or purplish-blue tint is seen in argyria and with prolonged ingestion of acetanilid

Cyanosis. The color is characteristically slate or purplish-blue and is sometimes more readily discernible in the subungual tissues than elsewhere in the body

Capillary Pulse. (See Chap. 8)

Nail-biting. This habit is generally regarded as an indication of poor emotional adjustment.

7

THE BREASTS, AXILLAE, AND BACK

THE BREASTS

Examination of the breasts must be performed systematically, with great care, and in a good light A casual glance and rapid passage of one's hands over these organs might well result in tragic oversight of an early malignant lesion. The two sides must always be compared. Especially if anything suggestive of carcinoma is encountered, this examination must be followed by careful search for enlarged lymphnodes in the axillae, neck and supra- and infraclavicular regions.

Normal breasts show great variation in size, shape, consistency, and the distance to which mammary tissue extends in various directions In some, the glandular element is much firmer than the surrounding fat and its distribution is readily determined by palpation. In others this distinction cannot be clearly made, their borders, especially the lateral and axillary, by shading into adjacent structures, defy sharp demarcation The breast may feel homogeneous and smooth, or lobular, nodular or lumpy. Sometimes, only the most experienced observer can distinguish between the normal and abnormal; for safety, surgical exploration of a suspicious area may be required

The developmental enlargement of puberty is likely to be accompanied by discomfort, tenseness, tenderness and perhaps appearance of striae Increased fullness or tenseness, and nodularity often occur with or without discomfort during the week before menstruation, subsiding with its onset; consequently re-examinations at different phases of the cycle are sometimes advisable. Pregnancy causes engorgement, enlargement and often striae, the glandular elements become more readily palpable During either puberty or pregnancy the changes do not necessarily start simultaneously on the two sides Following menopause the breasts become smaller and less firm due to atrophy of parenchymal elements and diminution of fibrous and fatty tissue

INSPECTION

Since certain variants are more easily detected in one position than another, inspection should be routinely performed with the patient erect, then leaning slightly forward from the waist, and supine Under pertinent circumstances, special maneuvers, as described below, are indicated. One looks for:

Asymmetry. Slight difference of size, shape or both is not infrequent as a

developmental anomaly and can be disregarded if other signs of trouble are lacking

Changes Incident to Pregnancy. (See Chap. 33.)

Flattening or Retraction of Nipple. Flattening or retraction must be distinguished from inversion, a uni- or bilateral phenomenon encountered in many normal women Here the nipple is depressed in a sulcus and can be expressed by gentle manipulation. The patient will state that the variant has existed since puberty. In carcinoma, traumatic fat necrosis, or as an aftermath of infection the nipple may become flattened, retracted, thickened or broadened Partial retraction is sometimes indicated by deviation of the direction in which it points, this may be brought out by pectoral muscle contraction as indicated below.

Erosion of Nipple. Erosion is sometimes concealed by a superficial crust and becomes evident only when the latter is removed.

Secretion from Nipple. If none is observed, one should attempt to express fluid from a duct by pressing in turn each segment of the areola downward toward the thoracic wall In the appropriate case if this procedure is unproductive, it should be repeated a day or so later after one or more of the ducts have had time to fill. Tight squeezing or rough manipulation is never in order. The implications of the various types of secretion are discussed below

Alteration of Color or **Texture of Skin.** Redness indicates increased circulation secondary to inflammation or to tumor, most likely cancer Especially in the upper segment the veins may be dilated A characteristic pocked ("orange-peel") appearance of the skin reflecting edema caused by blocking of lymph channels is an important sign of carcinoma, it may appear first on the areola

Cutaneous Puckering or Dimpling. Another important sign of carcinoma but sometimes seen in traumatic fat necrosis or following abscess, a local area of puckering or dimpling may be found over the lesion or a few centimeters beyond its margin It reflects pull on the septa of the breast by contraction of fibrous tissue within and around the lesion. This variant must always be looked for during routine examination with the patient leaning forward, upright, and recumbent If suspected carcinoma is not detected in this way, it might be demonstrable by one of the following maneuvers:

1 With the patient erect and one's hand under the breast, gently lift it upward

2. With her first upright, then recumbent, have her place her hands on her hips and press inward toward midline, a procedure which tenses the pectoral muscles

3. Have her lie with a pillow under her shoulder blades, head back on the table and arms outstretched above the head This is another method of tensing the pectoral muscles

other.





FIG 7 1 FIG 7 2

FIG 7 1 Dimpling of skin due to underlying carcinoma, brought out by manual elevation of left breast (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)
FIG 7 2 Chancre of nipple

In extensive carcinoma with fibrosis, pronounced shrinkage, distortion and cutaneous contraction will be at once apparent

Fixation to Pectoral Fascia. In early carcinoma this may be indicated by greater elevation of the diseased breast than of its mate when the pectoral muscles are tensed In the advanced case when the tumor is firmly fixed to the thoracic wall elevation will not occur Another procedure is to have the patient lean slightly forward from the waist with her chin up and arms outstretched horizontally Normally the two breasts will fall away equally from the body, if one is partially fixed, asymmetry of contour will be brought out.

Abnormalities Visible on Transillumination. With the patient in a dark room and a properly shielded light held against the skin and directed into the breast from various angles, one may normally identify near the nipple, dark cords representing ducts and vessels A cyst in the breast may appear as a circumscribed area of translucence, an adenofibroma as a circumscribed opacity, and a carcinoma as a less well defined opacity.

PALPATION

Using the fingers of one hand held together, one gently but firmly presses each segment of the breast in turn against the chest wall This should be done with the patient both erect and supine, as a rule, the latter is more satisfactory. When she is supine, each breast should be palpated while she is flat on the table, first with the homolateral arm at her side, then extended above her head If, as so often happens, the breasts droop laterally, each should then be re-palpated with a pillow under the homolateral shoulder but not under the head, a procedure which tips the trunk so that the breast on the supported side is flattened out on the chest This method reduces the chance of overlooking a

small lesion in the lateral zone which might be concealed by the thickness of the sagging tissue overlying it.

The temptation to palpate bimanually or with the thumb and fingers of one hand in opposition must be resisted since lobules of glandular tissue overlying each other might be wrongly interpreted as a mass. One exception to this rule is that, especially in the large, pendulous breast, palpation with the patient erect and the organ between the fingers of the two hands will occasionally reveal, particularly in the subareolar area, a small tumor which might otherwise escape notice. *Heavy pressure, hard squeezing, pinching or repeated palpations are of no value and must be avoided since they traumatize the patient emotionally and are thought to increase the hazard of disseminating metastases from a carcinoma* One feels for

Tenderness.

General Consistency.

Diffuse Nodularity.

Local Mass. Its size, consistency and sharpness of outline should be determined.

Fixation of a Mass. To determine fixation to an adjacent structure the examiner tests the mobility of the skin over the lesion and the mobility of the lesion itself within the breast and on the thoracic wall. This is best accomplished with the fingers of both hands or the thumb and opposed fingers of one hand.

Thickening or Fixation of Nipple.

Common Disorders

Congenital Anomalies. *Supernumerary breast elements, either one or more* rudimentary nipples on the chest wall below but in line with the breasts, or islands of mammary tissue laterally are not uncommon One or more painful nodules in or near the axilla, appearing during or shortly after pregnancy are most likely supernumerary mammary tissue; rarely, carcinoma develops in one of these islands Other congenital and developmental anomalies, such as absence of the breast or unilateral or bilateral hypertrophy, occur but are decidedly unusual

Painful Nodularity. Occurring most frequently in young women, especially accompanying or preceding the menses, this is often associated with minor abnormalities of the menstrual cycle The nodules are ill-defined, soft, elastic, tender, and ephemeral Characteristically they are multiple and occur in both breasts, especially in the upper, outer quadrants

Tender breast enlargement may precede by months the onset of other evidences of puberty in girls, occasionally exciting parental apprehension. Probably closely related are the firm, slightly enlarged tender breasts that sometimes occur in boys at puberty, often lasting for several months, to the embarrassment of the patient and the concern of his parents (see below)

Acute Mastitis. Most commonly seen soon after childbirth or early in lactation, this is indicated by redness, heat, tenderness, swelling, and often systemic evidences of inflammation Ordinarily this *caked breast* subsides spontaneously in a few days but may progress to suppuration (*breast abscess*), indicated by in-

crease of the above signs, fluctuation, and greater systemic reaction In rare instances, *non-suppurative mastitis* accompanies mumps.

Syphilis. CHANCRE may appear on the nipple, areola or skin of the breast; when it involves the nipple it can be mistaken for *Paget's disease*. The brief history, presence of early enlargement of axillary lymphnodes, relatively rapid changes in the local appearance, and discovery of *Treponema pallidum* on dark-field examination establish the diagnosis.

GUMMA of the breast is rare; it may be mistaken for carcinoma.

Tuberculosis. This is occasionally seen. It takes the form of indurated or broken-down, indolent, ill-defined, low-grade inflammatory areas, usually multiple, often presenting active or healed sinuses. Tuberculosis of an underlying costal cartilage may involve the breast secondarily, and retrograde infection from tuberculous axillary lymphnodes is possible.

Secretion from Nipple. Scant secretion—clear or cloudy, yellowish or milky—can be expressed from the breast in the newborn, during pregnancy, and, often, from the resting breast which has undergone repeated lactations This is bilateral. Unilateral, yellowish or clear secretion usually indicates cystic dilatation of a duct The dilated duct may be palpable as a localized, soft, oval swelling near the outer margin of the areola. *Secretion tending to form crusts about the nipple, especially if accompanied by local erosion, is strongly suggestive of Paget's disease of the nipple. Sanguineous discharge may be due to intraductal papilloma or to carcinoma.* Transillumination may disclose the intraductal papilloma as an oval or linear dark area near the areolar border.

Adenofibroma. Occurring chiefly in young women, this is a firm, elastic, freely movable, insensitive tumor, oval or slightly lobulated It is usually single, occasionally multiple.

Chronic Cystic Mastitis. Numerous pathologic classifications of this disease have been proposed but many observers believe that the various lesions described are all variants of the same fundamental disturbance. Commonly one finds a diffuse, usually insensitive nodularity of both breasts, often associated with one or more distinct, tense, rounded masses which may be inconstantly present. *Localized areas or single cysts may be indistinguishable from carcinoma without pathologic examination.* Transillumination may reveal the cysts as areas of rounded translucence, but such a finding does not exclude malignant disease, sometimes associated with the cysts. Distinction between chronic cystic mastitis and the functional disorders characterized by painful nodularity is also difficult, and sometimes impossible without exploration It is believed that up to the time of menopause, a woman with chronic cystic mastitis is much more likely to develop carcinoma than a woman whose breasts are normal; the likelihood of carcinoma developing after menopause in a woman with chronic mastitis is not much greater than in one with normal breasts

Galactocele. One or more circumscribed cysts appearing during or shortly after lactation are due to blocked ducts or to islands of breast tissue not connected with the duct system They are filled with a creamy secretion. They may be indistinguishable from tumor and require surgical exploration.

Traumatic Fat Necrosis. Scar tissue replacing interstitial hemorrhage and traumatized fat due to *injury may, through the* years, contract sufficiently to cause a mass with perhaps puckering, fixation, or retraction of overlying skin. This lesion may be easily confused with carcinoma.

Carcinoma. In its earliest stage, carcinoma cannot be distinguished from adenofibroma, a simple cyst, or the scar of traumatic fat necrosis. For this reason any single tumor of the breast should be explored and subjected to pathologic examination Carcinoma is usually hard, not sharply defined, and in its development tends to become adherent to the adjacent ducts, pectoral fascia or overlying skin Retraction, elevation or loss of erectility of the nipple points to involvement of the duct system. Dimpling, orange-peel appearance, edema or thickening of overlying skin, atrophy of the fat overlying the tumor, or demonstration of a pucker or drag when the mass is pushed to one side indicates adherence to the deeper cutaneous layers and usually means malignant disease

Later the breast becomes distorted, hard, increasingly fixed to the pectoral fascia and chest wall and, depending on the degree of fibrosis, either smaller or larger The skin becomes more fixed, puckered, infiltrated and eventually may ulcerate Isolated nodules of tumor tissue involving surrounding skin are common.

Even in the early stages careful search should be made for enlargement of regional lymphnodes in both axillae and the clavicular areas Pulmonary and hepatic metastases should also be looked for and x-rays taken of the thorax, spine, pelvis and cranium In advanced cases involved nodes become massive, fixed to the skin or thoracic wall, and sometimes ulcerate Edema of the arm indicates obstruction of lymphatic channels

Carcinoma recurring subsequent to mastectomy is indicated by nodules in the skin, perhaps with ulceration, extensive axillary changes, and often involvement of the other breast

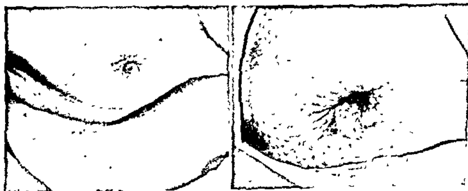


Fig 7 3 Fig 7 4

Fig 7 3 Retraction and elevation of right nipple due to carcinoma located just above areola at site of slight bulge (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)

Fig 7 4 Advanced carcinoma of right breast with enlargement, retraction of nipple, and orange-peel appearance of skin (Courtesy Pondville Hospital, Massachusetts Dept. of Public Health)



FIG 7 5 Massive carcinoma of right breast with invasion and ulceration of areola and nipple Retraction of skin laterally is also evident (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)

INFLAMMATORY CARCINOMA Although it may occur at any time, this is the type most likely to develop during pregnancy or lactation. The entire breast is larger, firmer and more tense than normal, a distinct mass is unusual The overlying skin may present an edematous and orange-peel appearance, and there may be a distinct red flush over the entire breast. Local warmth and tenderness are common, axillary lymphnodes become involved early The disease is highly malignant and runs a rapid course

PAGET'S DISEASE OF NIPPLE This is first indicated by crusting, erosion or ulceration, often accompanied by serosanguineous discharge Removal of crusts is likely to give rise to bleeding from the raw surface Tumor underlying the nipple, areola or surrounding skin may be too small to be felt A simple erosion or crusting area can sometimes be distinguished from Paget's disease by its prompt response to local ointments and antiseptics Any doubtful lesion must be regarded as cancer until pathologic examination has ruled it out. In the later stages there is progressive destructive ulceration of the nipple and areola, the underlying tumor becomes readily palpable. This form of carcinoma is highly malignant and accompanied by early regional lymphnode and remote metastases

Sarcoma. A relatively rare breast tumor, sarcoma is manifested by a rapidly growing, lobulated, freely movable mass It is firm but not as hard as carcinoma In contrast to the latter, which tends to show early adherence to skin or chest wall, sarcoma does not become fixed until late. At this stage, overlying skin is thin, tense, shiny and red but rarely ulcerates In spite of rapid local growth, sarcoma is less malignant than cancer. Axillary lymphnode metastasis is rare but remote metastasis, especially to the lung, is fairly common Pathologic examination is often required to distinguish sarcoma from a large adenofibroma.

Benign Enlargement of Male Breast Bilateral rounded prominence due to subcutaneous fat deposits is common in obese men and plump boys On palpa-

tion, the area feels relatively soft, smooth and homogeneous; *evidence of hyperplasia or disease of breast elements is lacking* Rarely, redness, tenderness and *swelling*, perhaps with suppuration, are encountered as a result of local infection.

MAMMAPLASIA. In the male adolescent, one breast, or both breasts simultaneously *or independently*, may show transient enlargement which disappears spontaneously after a number of months. The nipple and areola *increase in size* and beneath them, a well-defined, freely movable, often *tender mass may* be felt The latter represents hyperplasia of vestigial mammary elements and increased connective tissue akin to the changes in females at onset of puberty. The phenomenon may be observed in older men; here too the changes are likely to subside after a few months. In both adolescence and senescence the process is probably related to a shift in hormonal balance *It is not inflammatory* so that the term mastitis is a misnomer

Similar changes may occur at any time during life as a result of hormonal imbalance secondary to some systemic disorder. The most likely are testicular atrophy or neoplasm, adrenal cortical tumor, hypophyseal adenoma, hepatic cirrhosis, leukemia, lymphoma, pulmonary carcinoma, thyrotoxicosis, and malnutrition from any cause. Regression is the rule if the underlying disease is alleviated Intensive treatment with an estrogenic substance also causes mammaplasia which will disappear with cessation of therapy

GYNECOMASTIA Although often applied to cases of mammaplasia, this term, strictly speaking, should be reserved for those cases which, because of the nature of the underlying ailment, show no tendency to regress.

Carcinoma of Male Breast. One to two per cent of carcinomas of the breast occur in males. In contrast to cancer in the female, which may be deep-seated and hence not recognized until well-advanced, that in the male is *likely* to be apparent early because of the small size and shallowness of the breast elements The tumor is usually a painless enlargement with induration and retraction of the nipple, fixation to overlying skin and often to the deeper structures. Metastasis to axillary nodes may already have occurred by the time the tumor has attracted attention

THE AXILLAE

Hair. Scantiness of axillary hair is often observed in myxedema and other *endocrine disturbances, particularly those associated with gonadal* function. In the male with cirrhosis of the liver it is one of the signs of impaired estrogen metabolism. Pediculosis is not uncommon, especially in conjunction with pediculosis pubis The nits are dark and adhere to the hairs parallel to their longitudinal axes Parasites may be found, usually clinging to hairs close to the skin.

Skin. In the absence of overheating, excessive axillary sweating is most often a sign of nervousness It occurs in some persons without known cause. Pyogenic infection occurring in the form of furunculosis or deep abscess is frequently encountered Common cutaneous diseases are dermatitis, usually resulting from

sensitivity to deodorants, depilatories or clothing dyes, dermatophytosis often associated with dermatophytosis of the genital region, seborrheic dermatitis, and psoriasis

Enlarged Lymphnodes. Search for enlarged nodes is carried out with the patient upright and his arm relaxed and slightly abducted. The examiner, facing the patient, feels the left axilla with his right hand and *vice versa* The fingers, held loosely together, relaxed and slightly flexed, are introduced as high as possible into the axilla. They are then brought into contact with the thoracic wall and moved slowly downward along its surface toward the lower ribs. Nodes will be felt between the fingers and the wall. Next the procedure is repeated with the fingers of the other hand in contact with the medial surface of the upper arm, where nodes may be palpated by pressing toward the humerus In most normal axillae, a few small lymphnodes are palpable; they are of no significance Nodes which are larger, more numerous, tender, hard or soft, or appear fixed to skin or underlying axillary structure, usually indicate disease. The supraclavicular and infraclavicular regions must also be palpated, palpable nodes in these areas are abnormal and require investigation.

The common causes of axillary lymphnodopathy are:

1 Infection of the upper extremity or parts of the chest wall
2. Metastasis from cancer of the breast.
3 Diseases having generalized lymphnodopathy (see Chap. 5).

Blood Vessels. Changes similar to those found in the arm may be detected in the axilla.

THE BACK

Examination

Since some disturbances related to the back are more readily detected in certain positions than in others, the patient should, whenever possible, be examined standing, sitting and lying down Strain of the muscles or ligaments may exist independently or be secondary to disturbances in the bony framework. In fact, the predominating manifestations of bone or joint disease are often those of the associated muscular or ligamentous irritation, so that careful investigation is often necessary to differentiate between such diverse causes of back symptoms as postural strain, arthritis, posterior dislocation of an intervertebral disc, malignant disease of a vertebra, and some intrathoracic or intra-abdominal disease causing pressure or erosion *In any case of back injury or suspected bone destruction, caution must be used in having the patient move about and in testing his range of motion, until the extent of the damage has been determined by x-ray*

Back Pain. The pain may be sharp, take the form of a dull ache, or be deep and boring It may be restricted to a small area or involve a considerable part of the back, when it is acute, the patient may not be able to indicate its exact site Often it is referred along one or more extremities or around the trunk. Certain motions aggravate it, at times it makes motion impossible Back pain may have

its source not only in the spine or its supporting structures, but also in the spinal cord, nerve roots or a thoracic, abdominal, retroperitoneal or pelvic structure. When due to disturbance of the spine or supporting structures, it is often accompanied by stiffness which is characteristically worse following rest.

Posture and Gait. Routinely observed in the course of general physical examination (see Chap. 3), posture and gait sometimes require further study in relation to examination of the back. Normally if the skin over their tips is marked with a skin pencil, the spinous processes will fall in a straight line in the sagittal plane. Lateral deviation may be primary in the spine or its supporting structures, secondary to a skeletal variant elsewhere, such as a tipped pelvis due to inequality of leg length, or to some more remote cause such as unilateral pleuropulmonary fibrosis, obstructive atelectasis of a lung or inequality of muscle tone due to hemiplegia or poliomyelitis.

Except in early infancy when the back is straight in both the frontal and sagittal planes, it normally shows three anteroposterior curves. The cervical and lumbar segments are convex anteriorly, the thoracic curve, convex posteriorly. They merge into a single posterior convex curve on forward bending; on backward bending the converse is not true—the cervical and lumbar anterior curves are exaggerated, while the dorsal curve tends to flatten, but does not reverse. The manner in which these curves deviate from the normal is often helpful in differential diagnosis. For example, in poor posture all three curves are exaggerated. In acute sciatica from a local cause such as ruptured disc, the patient stands with a flat or reversed lumbar curve while the trunk lists forward and laterally; in degenerative arthritis of the hip, because of permanent flexion of the thigh, the pelvis is tipped forward so that in attempting to stand erect, the patient increases his anterior lumbar curve. Flattening of the lumbar curve is seen with spasm of the supporting muscles from any cause.

Abnormal gaits are described in chapters 3 and 36.

Swelling. A local swelling may be readily seen or felt in the upright subject, but if deep-seated, may be detected only by systematic, careful palpation, especially with the patient prone, the position in which the spinal muscles are most relaxed. Greater relaxation can be obtained by placing a pillow under the abdomen except when it is so prominent that extra support is unnecesary.

Cutaneous Lesions. If a person has been lying on his back, a blush or mottled redness due to heat, pressure or friction may be mistaken for an eruption or conceal or alter one which is actually present. Inspection of the skin should therefore be postponed until some minutes after a patient has shifted to some other position.

Tenderness. Palpation for points of tenderness is most effective when the patient is lying down; deep-seated tenderness may be brought out only when he is prone. Pain on pressure may be confined to a small area, as over a spinous process or sacro-iliac joint, or be diffusely distributed along one or more segments of the spine or supporting structures. One feels for tenderness over the spinous processes, the costovertebral angles, paravertebral muscles, lumbosacral

and sacro-iliac joints, sacrosciatic notches and the coccyx. Determination of the point of maximum tenderness is helpful in localizing a lesion.

Alteration of the bony landmarks may be detected while palpating for swelling and tenderness.

Muscle Spasm. When the patient is standing or sitting this may be seen as abnormal prominence of a muscle group or be felt as tautness and increased resistance List is usually present. These findings may not be apparent when the patient is lying down It is of little importance in the differential diagnosis of back disabilities, serving chiefly as substantiating evidence of local irritation.

Restriction of Motion. This may be noted at a glance but often can be brought out only by testing for range of movement. When restriction is pronounced, the patient may walk, stand, or sit bent forward or with a lateral list Ordinary movements are guarded To test for lesser degrees of limitation, the patient is examined standing, sitting and lying down; active and passive movements of each segment of the spine—cervical, thoracic and lumbosacral—are tested Due allowance must be made for age, body type, muscular development and habitus. Normally motion is freer in younger persons and those who are thin and lanky (For diagrams of tests for mobility, see chapter 35.)

Patient Standing. Test the cervical spine for flexion and extension of the head, rotation to right and left and lateral bending (see Chap. 5) Where restriction of motion is due to ligamentous or muscular irritation, lateral bending or rotation is likely to be the first motion impaired, in meningeal irritation, forward bending

Test the thoracic spine for flexion and extension and for rotation of the shoulders with the pelvis held fixed by the examiner. Lateral bending is a minor function of the thoracic spine To measure chest expansion, place a tape around the thorax just under the axillary fold and also at the lower border of the thoracic cage Normal expansion in the adult averages about 6 cm , it is less in thin, asthenic types and those with poor posture Expansion is diminished by any disturbance interfering with free respiratory excursion, such as spondylitis, barrel chest, pleuropulmonary fibrosis, asthma, pneumothorax, or inspiratory pain from any cause

Test the lumbosacral spine for flexion and extension and for side bending. Rotation is a minor function of this segment Flexion should be performed with the knees straight, normally, the lumbar curve reverses, becoming convex posteriorly and continuous with the thoracic curve. Forward bending is limited and reversal of the curve fails to occur in disorders of the lumbosacral spine or its supporting structures

Patient Sitting Compare the degree of forward bending with that observed in the standing position The hamstring muscles are taut when the patient stands, relaxed when he sits In disease affecting the lumbosacral spine, forward bending is essentially the same in the two positions. In muscular strain, sacro-iliac strain, and disorders of the pelvic girdle, it is freer in the sitting position

PATIENT LYING DOWN Ability to rise from the supine to the sitting position with the hands clasped behind the head is an index of the strength of the abdominal muscles. This movement also brings out pain in the muscles, ligaments, and joints of the back

Test passive straight-leg raising by placing one hand on the knee to prevent flexion at this point and raising the leg with the other hand, which is placed under the heel. The patient should remain completely passive. Note especially just where resistance is encountered Impairment is observed in any painful disorder of the lower spine or its supporting structures and is of value chiefly in indicating which side is more affected. Contracture of the hamstrings or gluteus maximus muscles or their surrounding fascia also limits straight-leg raising Test passive hyperextension of the thigh while the patient lies prone; lift the knee from the table, steadying the buttock with the other hand This may bring out pain in any of the low-back disorders In the presence of psoas abscess, passive hyperextension is restricted

While the patient is still prone, it is often convenient to test the ankle jerks (see Chap 36).

LOCAL SWELLING

Rheumatic Nodules. A common site of subcutaneous nodules of rheumatoid arthritis and acute rheumatic infection is along the course of the supraspinous ligament; they may be seen or felt in midline overlying the tips of the spinous processes These nodules are customarily painful only if situated at pressure points

Tumor. Lipoma and other forms of benign tumor are not uncommon; the former is especially likely to be present with spina bifida of the lower lumbar vertebrae or sacrum

Pilonidal Cyst. An anomaly of embryonic growth overlying the lower sacrum or coccyx, this may be manifested by nothing more than a dimple in the skin, nearby, in midline, a soft, circumscribed swelling 0 3–2 cm in diameter is sometimes palpable in the subcutaneous tissue Both cyst and dimple may go unnoticed until secondary infection, common in early or mid-adult life, supervenes The resulting abscess can be felt as a tender, indurated subcutaneous mass, it eventually discharges externally near the upper part of the gluteal fold in or near midline (*pilonidal sinus*) The discharge, which may be continuous or intermittent, is apt to contain hair as well as purulent material and detritus

Other congenital cysts are also found in the sacral region, like pilonidal cyst, most have no communication with the spinal canal Their nature can be determined only by surgical and pathologic means

Meningocele. This anomaly is a congenital, saccular swelling representing a herniation of the dura, arachnoid and spinal nerve roots through a *spina bifida* —a developmental defect in the laminae and spinous processes of the vertebrae or sacrum. It may occur at any point between the occiput and sacrum but in 90 per cent of cases is in the lowest third of the spine. It is often translucent; a tuft

of hair is frequently present on the overlying skin. It is fluctuant and may expand slightly on straining. On deep palpation one notes absence of spinous processes in midline underlying the tumor. In an appreciable number of cases, herniation of part of the spinal cord also occurs (*myelomeningocele*), resulting in some form of paralysis, the character of which depends on the level of the lesion and the amount of nerve tissue involved. Occasionally, meningocele occurs through a defect in the midline of the skull.

Spina bifida often exists without signs or symptoms (*spina bifida occulta*) A suggestive indication sometimes found is an abnormal patch of hair somewhere along the spine The bony defect may be discovered accidentally on routine palpation or x-ray examination

Perinephric Abscess. When it reaches the surface, the infection causes fullness, redness, acute tenderness, and later, definite swelling in the flank somewhere between the crest of the ilium and the twelfth rib.

Tuberculous Abscess. Cold abscess originating in a tuberculous vertebra or retroperitoneal lymphnode may point in the lumbar region as a slowly developing, relatively painless swelling As a rule however, it follows down the sheath of the psoas muscle and points in the femoral triangle below Poupart's ligament; less often it appears in the iliac fossa or the gluteal region. Tuberculous abscess starting from a necrosed rib is occasionally seen higher in the back. Actinomycosis may present a similar picture.

Appendiceal Abscess. A suppurative process arising from retrocecal appendicitis may extend posteriorly and cause tenderness, redness and fullness or swelling in the right flank

Aneurysm of Descending Aorta. Pulsation or swelling may be found in the back near the angle of the left scapula (see Chap. 17).



Fig 7 6 Spina bifida of cervical spine with meningocele

Cutaneous Lesions

Most of the skin lesions discussed in chapters 3 and 8 may be found on the back as well as on the front and sides of the thorax.

Decubitus Ulcer. Bedsore occurs as a result of local pressure, poor local circulation, and irritation in bedridden patients whose skin has not received adequate nursing care. Emaciation, debilitating disease, and neurotrophic disorders are often contributing causes. It is found over bony prominences, especially the sacrum, trochanters and heels. Beginning as a small area of redness, it spreads rapidly and, as a result of secondary infection, breaks down within a few days, producing a foul, sloughing ulcerative lesion involving the skin and deeper structures. Decubitus ulcer can usually be prevented by proper nursing. *Infection deeply underlying such an ulcer may be the cause of sustained fever otherwise unexplained. It is the duty of the physician whenever a patient is confined to bed—especially if he is debilitated—to be on the alert for the first sign of beginning decubitus ulcer.*

Disorders of the Spine and Supporting Structures

Scoliosis. Lateral curvature is a deformity in which a series of vertebrae show lateral deviation accompanied by some degree of rotation.

Functional. The curve is usually C-shaped and involves chiefly the thoracic segment with convexity almost always to the left and with minimum rotation of vertebral bodies. A high shoulder, asymmetrical scapulohumeral angles, and prominence of one iliac crest are usually present. When of slight degree, the de-



Fig. 7 7 Extensive decubitus ulceration.

formity can be detected only by marking with a skin pencil the line made by the spinous processes. In contrast to structural scoliosis, the curve disappears on forward bending, and with the patient erect, can often be voluntarily corrected The usual causes are faulty posture, general muscular weakness, or inequality in length of the lower extremities. If untreated, it may in time progress to the structural type.

STRUCTURAL. There is usually a primary thoracic curve with compensatory curves above and below, creating an *S*-shaped deformity. Here distortion is due to definite skeletal alterations so that the deviations cannot be corrected voluntarily by the patient The vertebral bodies are rotated toward the convexity, the spinous processes toward the concavity The thorax is consequently deformed with the ribs flattened anteriorly and prominent posteriorly on the side of the convexity On forward bending the curvature remains and the posterior prominence of the ribs becomes more apparent Asymmetry of shoulder levels and greater prominence of one hip also occur. The thoracic deformity and consequent impairment of ventilation may be sufficent eventually to cause pulmonocardiac failure (see Chap. 27) Structural scoliosis may be congenital or develop before adolescence from unknown cause. It also results from poliomyelitis, disease or injury of the vertebrae such as rickets, osteomalacia, tuberculosis or fracture, long-standing functional scoliosis, and such causes as chronic pleuropulmonary disease causing asymmetrical contraction of the thoracic cage, deformities of the shoulder, or inequality in length of the lower extremities

Kyphosis. This is exaggeration or angulation of the normal posterior curve,

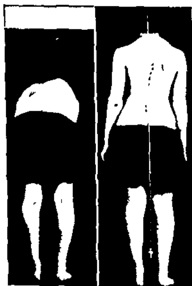


FIG 78



FIG 79

FIG 78 Structural scoliosis Note prominence of right ribs when trunk is bent forward.
FIG 79. Congenital kyphosis

or posterior angulation of a normally anterior curve Angulation or a sharp curve usually means tuberculosis, syphilis, malignant disease, congenital vertebral anomaly, or compression fracture. A gradual curve, seen almost always in the thoracic region, is most commonly due to faulty posture, general weakness from any cause, congenital deformity, occupational stooping or load-carrying, senile osteoporosis, chronic arthritis of the spine, rickets, Scheuermann's disease, hypertrophic emphysema, paralysis agitans, Paget's disease, acromegaly, or hyperparathyroidism

Occasionally a combination of scoliosis and kyphosis occurs; in these cases the likelihood of eventual cardiopulmonary disease is greatest.

Lordosis. One of the forward curves of the spine, usually the lumbar, is exaggerated The most common causes are faulty posture, weakness of the back muscles from any cause, enlargement and excessive weight of the abdomen, as in ascites or pregnancy, congenital dislocation of the hip, flexion contracture of the hip as in tuberculosis or arthritis, forward displacement of a vertebral body, compensation for kyphosis above or below, and some types of abnormal skeletal development.

In scoliosis, lordosis and kyphosis, pain, as a rule, is not outstanding except when there is associated active disease or unusual strain produced by the mechanical disadvantage.

Back Strain. Acute strain is brought about by direct trauma, some sudden or unaccustomed movement such as sneezing, twisting or heavy lifting, or persistent use of a chair or bed which does not give proper support to the lower segment Chilling, exposure to a draft, fatigue and poor posture are often contributing factors Pain is pronounced and aggravated by motion, in severe cases,



Fig 7 10 Marked lordosis of lumbar spine resulting from anterior poliomyelitis

list and marked restriction of movement are present Local tenderness is not necessarily striking but can usually be elicited in a limited area.

CHRONIC strain results from repeated or prolonged acute strain or from mechanical disadvantage derived from habitually faulty posture or a skeletal deformity. Indications may be entirely subjective—backache and stiffness—or one may find postural deformity, tenderness, moderate restriction of motion and perhaps muscle spasm

Arthritis. (See Chap. 35.)

Tuberculosis. (See Chap 35.)

Infectious Processes. Osteomyelitis, typhoid fever, brucellosis, and other bacterial and fungus diseases may be accompanied or followed by backache and other indications of spinal column involvement. The diagnosis depends on the general clinical picture, laboratory findings and x-rays.

Malignant Disease. Primary or metastatic malignant disease of the spine is characterized by pain, often referred around the trunk or along an extremity, local tenderness, muscle spasm, and sometimes, in the later stages, kyphotic angulation secondary to spontaneous compression fracture In any obscure case of back pain, malignant disease must be considered. Symptoms are often present before vertebral changes are apparent by x-ray. Manifestations of metastatic disease in the spine may precede those of the primary lesion.

Fracture. Compression fracture, seen most commonly in the thoracolumbar region, is indicated by acute pain aggravated by movement, local tenderness, spasm and impaired motion The spinous process of the injured vertebra is prominent and acutely tender on pressure and percussion. If improperly treated, the symptoms may disappear spontaneously, only to recur later as a result of chronic strain due to the permanent mechanical handicap. When, as sometimes happens, the posterior aspect of the vertebral body protrudes into the neural canal, neurologic changes appear, their extent depending on the degree of cord damage. Spontaneous fracture, such as is encountered in senile osteoporosis, may show minimal symptoms or none, sometimes the lesion is discovered incidentally by x ray. In fracture of a transverse process, tenderness is, as a rule, limited to the affected side of midline and there is no deformity. Rare fractures are those of a spinous process or articular facets.

Lumbosacral Strain. Due to irritation of supporting structures, this common ailment may be initiated by acute abnormal stress, such as heavy lifting, or a fall or near-fall, or it may have a more insidious postural or other mechanical origin. In the *acute* stage, one finds low-back pain often referred uni- or bilaterally to the gluteal region and sometimes to the posterior and lateral aspect of the thigh, posterolateral aspect of the calf, and outer side of the foot List to one side, flattening or reversal of lumbar curve, muscle spasm, limitation of back motion, and regional tenderness with perhaps tenderness over the sacroiliac joint and in the sacrosciatic notch are present. Forward bending is restricted when standing, sitting or lying. Active and passive straight-leg flexion and perhaps straight-leg hyperextension are painful Hip motions are free. In the *chronic* stage the findings are essentially the same but less pronounced.



FIG 7 11 A Spondylolisthesis, lateral view Fifth lumbar vertebral body has slipped forward in relation to sacrum with resultant forward displacement of spine above Note exaggerated angulation at lumbosacral articulation Arrow points to bony defect of neural arch of fifth lumbar vertebra
B Normal spine for comparison with A

Arthritis, pyogenic infection, tuberculosis, and tumor, usually metastatic or invasive, may, by involving the lumbosacral region, give a picture closely resembling severe strain

Spondylolisthesis. Due to a developmental defect between the pedicles and laminae, the body and anterior part of the arch of the fifth lumbar vertebra are *displaced forward carrying with them the spinal column above* Less commonly, the fourth lumbar vertebra is affected and slips forward on the fifth Symptoms which are precipitated either by sudden trauma or chronic strain are stiffness and dull, aching lumbosacral pain, often radiating down one or both extremities and aggravated by exercise or strain Forward bending is limited, lateral *movements free The lumbar curve is angulated. The spinous process of the* affected vertebra which, by virtue of the nature of the defect does not slip forward, may be seen or felt as a local prominence with a tender depression above The trunk appears shortened, with the ribs unusually close to the iliac crest

Sacro-iliac Strain. The ligamentous supports of the sacro-iliac joints are so *strong that strain is unlikely except when trauma is particularly severe.* Many cases of back and extremity pain which were formerly attributed to sacro-iliac

strain are currently believed due to some lumbosacral disorder or protruded intervertebral disc. The sacro iliac joint is subject to the same diseases as those which affect the lumbosacral region The manifestations are virtually the same as in lumbosacral trouble, except that back motions are likely to be less restricted when the patient is sitting. X-ray demonstration of haziness with later narrowing or obliteration of one or both sacro-iliac joints is an important confirmatory sign of early rheumatoid spondylitis.

Posterior Protrusion of Intervertebral Disc. Severe strain, as in lifting a heavy load or injury such as occurs in a slip, fall or automobile accident, is the most likely cause. *In about 50 per cent of cases no history of previous trauma can be elicited.* Since over 90 per cent of protrusion causing trouble occurs in the low back, the incorrect diagnosis of sacro-iliac or lumbosacral strain is often made. Conversely, ankylosing arthritis of the low back may produce a clinical picture readily confused with a disc lesion.

Lumbar Disc The most prominent symptom is recurrent, often incapacitating attacks of low-back pain, with associated severe sciatic pain extending down the back of the thigh and outer side of the calf. Aggravation of the sciatic pain by cough, sneeze or jugular vein compression is significant. A small lateral rupture may give back pain only Restricted straight-leg raising and forward bending in both the sitting and standing positions, local tenderness, flattening, stiffness and perhaps scoliosis of the lower back, are the outstanding signs Diminution or absence of ankle jerk and sometimes segmental paresthesia or



Fig 7 12



Fig 7 13

Fig 7 12 Myelogram showing filling defect created by posterior protrusion of intervertebral disc between fourth and fifth lumbar vertebrae

Fig 7 13 Prominent scapula due to paralysis of serratus magnus muscle.

anesthesia of the homolateral extremity may be observed Occasionally increased ankle jerk is found. In the later stages, atrophy of the gluteal, thigh or calf muscles often develops

CERVICAL DISC. The outstanding manifestations are pain in the neck and, depending on the disc involved, pain, paresthesia, weakness, atrophy and twitching of muscles in the scapular region and areas of the arms and hands supplied by the affected nerves. Pain is usually sharp, stabbing and often increased by motion of neck or arm Deep arm reflexes may be diminished. Evidence of homolateral corticospinal tract disturbance—increased knee and ankle jerks, Babinski response—below the site of the lesion may be found.

THORACIC DISC. Protrusion of a disc in the thoracic region is rare.

Total protein of the spinal fluid is usually moderately elevated As a rule, diagnosis can be confirmed by x-rays taken after injection of radiopaque medium into the spinal canal (*myelography*), a filling defect created by the protruding disc will be seen Sometimes in the equivocal case, injection of contrast material directly into the suspected disc (*discography*) might be indicated With this procedure, one may find changes consistent with the diagnosis of protrusion.

Coccygodynia. (See Chap. 34)

THE SCAPULAE

Although part of the shoulder girdle, the scapulae are usually examined along with the back.

Prominence. When *bilateral*, this is usually due to poor posture or exaggeration of the thoracic curve of the spine from some other cause. It is also observed in atrophy of related muscles When *unilateral*, it is secondary to lateral curvature of the spine or to paralysis of the serratus magnus muscle Paralysis of this muscle can be recognized by unusual prominence of the scapula brought out by having the patient push forward, against resistance, with the homolateral hand

Elevation. Congenital elevation of the scapula (*Sprengel's deformity*) is usually unilateral. It is relatively rare and almost always associated with congenital deformity of the spine On raising the homolateral arm, the scapula fails to move laterally.

Tumor. Tumor of the scapula is uncommon

OSTEOCHONDROMA appears as a rounded, non-tender, bony swelling

SARCOMA, occasionally encountered in children, is rare after the second decade. In a child, a solid, virtually painless tumor of the scapula always suggests this disease but it may be osteochondroma

Fracture. Fracture of the glenoid cavity or body is the most likely The former is usually due to a direct blow on the outer aspect of the shoulder; the latter as a rule is comminuted and due to direct blow over the shoulder blade itself Pain, local tenderness, swelling, and restriction of shoulder movement are present.

INSPECTION OF THORAX

REGIONAL ANATOMY

The positions of structures within the thorax and the local signs to which they give rise are indicated with reference to certain landmarks on the thoracic wall These are the clavicles, sternum and ribs, anteriorly and laterally, and the scapulae, vertebrae and ribs posteriorly. Thus we speak of rales as heard "above the left clavicle anteriorly," "below the right scapula," "between the seventh and ninth ribs in the axilla," and so on Position in the frontal plane can be indicated in relation to a line drawn perpendicularly through the nipple (*nipple line*) or one dropped from the anterior fold, posterior fold or apex of the axilla (*anterior, posterior and midaxillary lines*) Because of the variability in tenseness of the skin and subcutaneous tissue, position and size of the breasts, and thickness of subcutaneous fat in different subjects, these lines are not dependable for accurate measurements

When exactness is required, points in the frontal plane are best located by measuring from midsternal line, midline of the back or midclavicular line. The last-named lies midway between the midsternal line and one dropped vertically downward from the outer end of the clavicle The size and position of the heart, for example, are ascertained by determining the location of the apex impulse and lateral borders in relation to midsternal and midclavicular lines

One must be familiar with·

1. Position, with reference to the bones of the thorax, of the heart, various lobes of the lungs, liver and spleen

2. Percussion outlines of these structures Because the heart, liver and spleen are partially overlain by air-containing lung tissue, their percussion outlines do not exactly correspond to their actual borders However, there is a fairly constant relation between them and actual size so that size and position can be estimated with considerable accuracy by careful percussion (see Chap 10).

3. So-called valve areas of the heart—the points at which sounds originating in the valves are best heard These do not correspond to the anatomical sites of the valves

Since most of the stomach lies behind the ribs and left lobe of the liver, only a small portion of this organ is normally directly accessible to physical examination The kidneys are frequently so; on the other hand, in many persons,

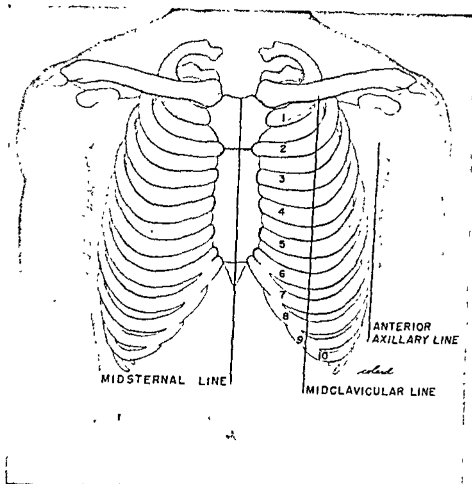


Fig 8 1 Location of reference lines on chest wall

particularly those with long, narrow builds, the right, and rarely, both kidneys, may be felt.

THE THORAX AS A WHOLE

Size and Shape

The size of the thorax in normal persons is variable. A small thorax is most likely to be seen in a patient who has been long bedridden or who has suffered in infancy from rickets, hypertrophied adenoids, or a combination of the two. An abnormally large thorax (*barrel chest*) is most often encountered in connection with structural skeletal changes and the hypertrophic type of emphysema.

The thorax of a child is more nearly cylindrical than that of the adult; the

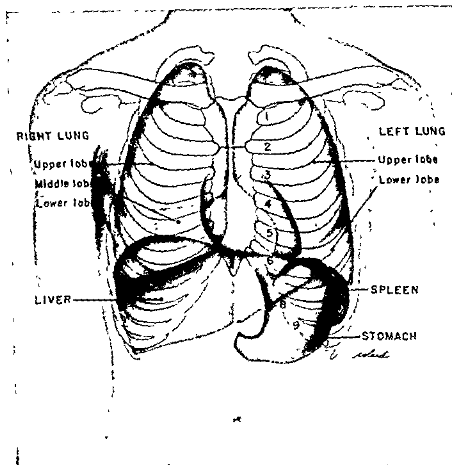


FIG 8 2 Position of heart, lungs, liver, stomach and spleen.

anteroposterior diameter almost equals the lateral. The adult thorax is at first flattened, the lateral diameter being appreciably greater than the anteroposterior; with advancing years it tends to return to the childhood shape. In early years the commonest pathologic modifications are due to hypertrophied adenoids or rickets, in middle and later life, to osteoporosis, phthisis, hypertrophic emphysema or other chronic pleuropulmonary disease, or to some spinal deformity With emaciation the ribs, scapulae and clavicles become prominent, without actual change in shape of the thorax. Tuberculosis of the pulmonary apices may produce retraction of the tissues above and below the clavicle without emaciation

Rachitic Chest. The sternum, especially its lower half, usually projects and the sides of the thorax are compressed, sloping in to meet it as the sides of a ship slope down to meet the keel (*pigeon breast*). In other cases the lower part

of the sternum is depressed (*funnel chest*) Rickets also causes a depression of the thoracic wall which extends from the ensiform cartilage downward and outward toward the axillae, following the course of the diaphragmatic attachment (*Harrison's groove*). The lowest portion of the cage anteriorly flares outward, owing to enlargement of the liver and spleen beneath and the pull of the diaphragm above. Along the lines of the chondrocostal articulations may be felt, and sometimes seen, a row of bead-like swellings (*rachitic rosary*) Pigeon breast and other deformities are sometimes congenital.

Flat Chest. The normal anteroposterior flattening is exaggerated, the shoulders stooping, the clavicles and scapulae prominent, the neck generally long, the costal angle acute. Although this is the popular concept of the tuberculous thorax, actually it is most commonly seen in persons with poor posture with or without faulty nutrition. It is also encountered in extensive bilateral pleuropulmonary fibrosis

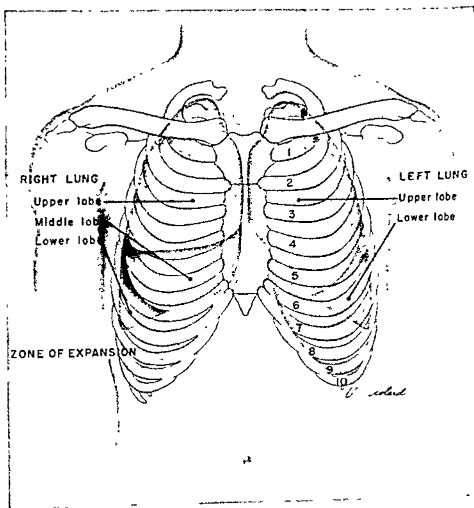


FIG 8 3A

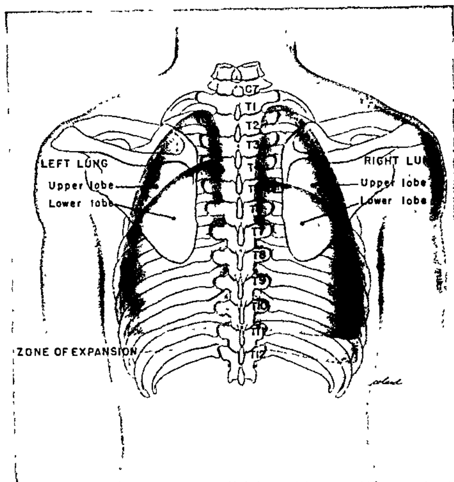


FIG 8 3B

FIG 8 3. Position of pulmonary lobes A Anterior B Posterior

Barrel Chest. Seen especially in males, the thorax is enlarged, particularly by increase of anteroposterior diameter It is usually associated with hypertrophic emphysema or long-standing bronchial asthma, but may be seen without either.

DEFORMITIES

In contrast to the variants just enumerated, which are symmetrical, those described below chiefly affect particular portions

Distortion due to Spinal Disease. Moderate or extreme structural scoliosis causes asymmetry of the thorax and often displacement of intrathoracic organs The distortion makes the usual bony landmarks unreliable, the apex of the heart may be found in the fourth space or in the axilla, and portions of the lungs may be compressed In thoracic kyphosis, shortening of the trunk and in-

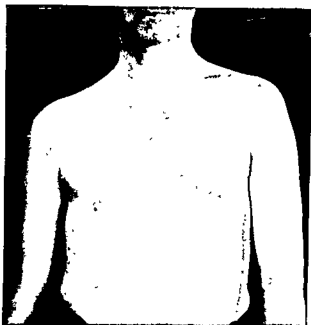


FIG 8 4 Flattening of right side of chest due to chronic fibrous tuberculosis of underlying lung and pleura

crease of the anteroposterior diameter sometimes cause prominence of the upper chest

Unilateral Flattening. In well-established unilateral pleuropulmonary fibrosis or obstructive atelectasis, contraction of the affected side of the chest may be seen The shrinkage is made more obvious by contrast with the sound side, which may become prominent because of compensatory hypertrophy of the unaffected lung

Unilateral Prominence. In large pleural effusion or pneumothorax, less often in pulmonary or pleural malignant disease, the affected side may appear increased in size The depressions between the ribs tend to be smoothed out.

Local Prominence. The most common causes of local prominence are

1 Greatly enlarged heart or pericardial effusion, especially in children In adults precordial prominence may be seen as a result of cardiac enlargement occuring in youth or occasionally as a normal variant.

2. Deformity due to scoliosis

3. Kyphosis

4. Large aneurysm of the ascending segment or arch of the aorta.

5. Tumor of chest wall, lung, or mediastinum

6. Cold abscess (tuberculosis, actinomycosis) of a rib or the sternum.

7. Empyema perforating the chest wall (*empyema necessitas*)

8 Subcutaneous emphysema. The chest wall has a swollen or puffy appearance, localized at first, but rapidly becoming more widespread. On palpation, characteristic crepitus is felt beneath the skin

CARDIOVASCULAR MOVEMENTS

The Normal Apex Impulse

In most persons one can see (and feel), synchronous with cardaic systole, an outward movement of a small portion of the left chest wall caused by the beat of the cardiac apex. This is the cardiac or apex impulse It should be studied with the patient upright and lying in various positions. Since, in many instances, this impulse may be fairly diffuse, *it is best for practical purposes to consider the center of maximum movement as the apex impulse, although this does not exactly correspond to the actual apex of the heart, which is somewhat further outward and downward* It is more easily seen in people with thin chest walls In some normal persons, in those who are obese or have thick chest walls, and in women with large or pendulous breasts, the impulse may not be visible This may be true, too, in certain pathologic conditions within the thorax, such as pleurisy with effusion, pericarditis with effusion, or emphysema

The apex impulse should not be confused with the systolic retraction often seen, particularly in thin subjects, over a somewhat wider area between the sternum and the apex. This is due to falling away from the chest wall of the right ventricle as it contracts and the heart rotates

In adults, the usual position of the maximum impulse is the fifth intercostal space on or just inside the midclavicular line Less often, it may be in the fourth interspace or under the fifth or sixth rib In normal infants, young children, and persons with high diaphragms, the impulse is apt to be in the fourth space and somewhat further laterally. In the asthenic type of person with long, narrow thorax and low diaphragm, it may be found as low as the sixth intercostal space Its distance from midline (a measurement which should be taken *tangentially* from midsternal line, *not circumferentially* around the chest) is normally 7–9 cm, varying with the size of the individual.

If the sitting or standing subject bends his trunk well to the left, the impulse [illegible]

to move in this manner is sometimes of diagnostic significance in certain intrathoracic diseases

Such factors as thickness of the chest wall, status of the lungs and pleura, size of the heart and its individual chambers, and the force of its beat must be taken into account in judging whether or not the apex impulse is abnormal

The impulse of the normal heart ordinarily covers an area 2–3 cm. in diameter, its left margin coincides with the left cardiac border. In an enlarged heart or one whose action is particularly vigorous from some other cause such as exertion or thyrotoxicosis, it may be more forceful and widespread Obviously it will be more evident or diffuse in a thin-chested person or in one whose lungs cover less of the heart surface than is usual, as in chronic pulmonary fibrosis on the left.

THE ABNORMAL APEX IMPULSE

Increased Force. Increased force and a widened area of impulse are encountered when the heart is overactive or hypertrophied. Because it tends to rotate as it contracts, one often observes, in marked hypertrophy, retraction in one area with an outward impulse elsewhere, giving a rocking motion to the chest wall Here a point of maximum impulse roughly corresponding to the apex can usually be determined.

Diminished Force. In acute cardiac dilatation, the apex impulse, if visible or palpable, is diffuse, feeble and whipping in character Cardiac action may be so feeble that no impulse can be seen or felt.

Abnormal Position. Common causes of displacement of the cardiac impulse other than those effected by the normal variants mentioned above are:

HYPERTROPHY AND DILATATION As the heart enlarges, its apex tends to move outward and downward, perhaps reaching the sixth or seventh intercostal space or as far out as midaxillary line In general, left ventricular hypertrophy tends to move it downward and outward, right ventricular hypertrophy, outward but not usually appreciably downward In the latter, however, pulsation may be seen to the right of midclavicular line as a result of systolic impact of the large right ventricle against the chest wall.

ABDOMINAL ENLARGEMENT Pronounced distention of the adbomen, as by gas, fluid, pregnancy, or large tumor, pushes the impulse upward and to the left

PLEURAL EFFUSION OR PNEUMOTHORAX Fluid or air in the left pleural cavity, when present in a considerable quantity, will displace the heart toward the right Depending on the amount present, the impulse may be just to the left of the sternum, concealed by it or, in extreme cases, to the right of it Air or fluid in the right pleural cavity will shift the heart toward the left, but the effect is not so pronounced, an appreciable alteration is noticeable on clinical examination only when a large amount of fluid or air is present

RETRACTION OF LUNG. In severe unilateral pleuropulmonary fibrosis or obstructive atelectasis, the apex impulse moves toward the affected side

DEFORMITY OF THORAX The heart and other intrathoracic structures are forced out of normal position in cases of spinal curvature, funnel chest, or other skeletal deformity

DEXTROCARDIA OR VISCERAL TRANSPOSITION

VISCEROPTOSIS When this is pronounced, the heart may be low in the thorax so that its apex impulse is found in the sixth space or below, without lateral deviation Rarely, downward displacement is encountered in arteriosclerosis as a result of stretching of the aorta, in aneurysm, or in mediastinal tumor.

APICAL RETRACTION

Visible retraction of that part of the lower precordium lying just to the left of the sternum is not unusual with the beat of the normal heart, particularly in a thin person In pronounced right ventricular hypertrophy, one may observe an outward systolic impulse over midprecordium with simultaneous retraction in

the apical region. Systolic retraction is often wrongly interpreted as an important indication of adhesions between the pericardium and chest wall. As a matter of fact, it is not found in most of these cases, although theoretically it should occur from the pull of the cardiac apex

Broadbent's Sign. Although not, strictly speaking, an apical phenomenon, this sign can properly be included in a discussion of the apex impulse. The term refers to systolic retraction of the intercostal space which is occasionally seen just below the angle of the scapula or in the postaxillary line at the same level Broadbent considered it an important sign of fibrous pericarditis with adhesions to adjacent structures, but actually it is not always seen in this disease Furthermore, it sometimes occurs in normal persons, particularly those with thin, elastic chests, and in patients with cardiac hypertrophy. Its diagnostic significance is relatively slight.

Other Pulsations

Epigastric Pulsation. Pulsation in the epigastrium is frequently seen The movement may be a retraction synchronous with and related to cardiac systole, or a forward impulse transmitted from the abdominal aorta and occurring just after systole It occurs in some perfectly normal people, in others it is brought out by overaction of the heart as from extra exertion or excitement It has little value in the diagnosis of heart disease Less commonly, epigastric pulsation may be visible as a result of pulsation of the liver in cases of marked venous congestion usually, but not always, with tricuspid valve insufficiency. Rarely, it may be due to abdominal aneurysm or be transmitted from the aorta by a high intra-abdominal tumor

Suprasternal Pulsation. Sometimes seen in normal persons, this is common with overactivity of the heart from any cause and with aortic dilatation, aneurysm, or arteriosclerotic dilatation of the carotid arteries Pulsation above the inner halves of the clavicles reflects arteriosclerosis of the subclavians

Aneurysm. Local pulsation from an aneurysm may appear in almost any part of the chest, usually in the second or third right intercostal space near the sternum Sometimes it is seen in the back, usually on the left side and above the fourth rib Before the aneurysm has perforated the chest wall and appeared externally as a tumor, the movements transmitted to the wall may be slight, being seen only on painstaking inspection. In searching for slight pulsations, the observer, his eyes level with the surface of the chest, should look across it toward the light, against which the chest is silhouetted *When local pulsation is seen in the upper intercostal spaces, it is much more commonly due to vigorous heart action or retraction of a lung than to aneurysm, and its diagnostic value is limited*

Coarctation of Aorta. Compensatory enlargement of the internal mammary or intercostal arteries may cause visible (or palpable) pulsation along their courses.

Pulsating Purulent Pleuritis. Very rarely, pus in the pleural cavity will burrow through the intercostal spaces and cause a local tumor on the chest wall, usually on the anterior or lateral aspect between the third and sixth interspaces



FIG 8 5 Inspection of chest wall for local prominence or slight pulsation Examiner looks toward the light across body surface (One sights across abdomen in a similar manner when looking for slight prominence, pulsation, or movements reflecting peristaltic activity, as described in Chapter 29)

Such collections of pus may occasionally transmit the cardiac impulse and pulsate, but this is most uncommon.

RESPIRATORY MOVEMENTS

NORMAL BREATHING

In normal respiration the ribs move outward and upward with inspiration, downward and inward with expiration The lungs fill the thoracic cavity and are prevented from collapsing by the negative intrapleural pressure The thorax expands by muscular action and falls back passively with expiration, which normally endures slightly longer than inspiration. Paradoxically, as will be noted in the discussion of auscultation of the lungs (see Chap 11), the normal breath sound produced by inspiration is longer than that of expiration

The normal rate is 14–18 per minute, slightly higher in females In the newborn it averages 44, gradually slowing as the child grows older Since the diaphragm performs more of the respiratory work in men and children, movement of the lower thorax and upper abdomen is more pronounced In women, the more important role of the intercostal muscles emphasizes upper thoracic movements.

CHANGES IN EXPANSION

If the patient is observed while breathing normally or forcefully, certain variations from the normal type of respiratory movements may be seen Variations in expansion, if present, can sometimes be better observed by adopting the following procedure:

Spread the fingers of both hands and place them on the two sides of the upper

chest, the forefinger below the clavicle and the other fingers on the successive ribs. Instruct the patient to breathe deeply If the chest is of the normal, flexible type, the fingers will rise one after another, beginning with the forefinger. They may rise simultaneously, indicating a senile or emphysematous chest. Limitation of expansion on one or both sides is indicated by only slight elevation of the fingers; increased expansion, by exaggerated elevation.

Diminished Costal Breathing. Accompanied by compensatory increased diaphragmatic excursion, this can be brought about by such causes as the pain of acute pleuritis, chronic pulmonary fibrosis, fixation of the ribs due to spinal arthritis or costochondral calcification, and paresis of the intercostal muscles, as in poliomyelitis with high spinal cord involvement. In such instances, the pull of the diaphragm on the lower ribs can sometimes be seen.

Increased Costal Breathing. This occurs as a compensatory mechanism in disturbances limiting downward movement of the diaphragm, such as ascitic fluid, enlarged liver or spleen, large abdominal tumor, pain of acute peritonitis or diaphragmatic pleuritis, or paresis of the diaphragm, as in bulbar palsy.

Diminished Unilateral Expansion. This may be due to pleural effusion, pneumothorax, or solid tumor of lung or pleura; the affected side, in such instances, may seem fuller than normal. Or, expansion may be reduced, as in chronic pleuropulmonary fibrosis or total occlusion of a bronchus. The side then appears retracted. Restricted motion on one side may also be due to pain, usually of pleural origin, or to pressure upward from below the diaphragm, less often to unilateral phrenic paralysis Tuberculosis of one apex may cause restriction of motion in the upper part of the corresponding side, probably as a result of lessened pulmonary elasticity.

Increased Unilateral Expansion. Overactivity of one lung compensates for reduced activity of the other.

Abnormal Breathing

Dyspnea. In its strictest sense, this term refers to the subjective sensation of difficulty or distress in breathing It is also used in an objective sense to include all degrees of difficulty, from simple shortness of breath to persistently labored breathing which calls into play muscles not ordinarily employed in respiration

Numerous factors, mechanical and chemical, are involved in the production of dyspnea Usually they act in conjunction, occasionally singly. Cyanosis, due to diminished oxygenation of arterial blood, frequently accompanies dyspnea, especially in congestive heart failure, advanced pulmonary disease, and mechanical obstruction to flow of air However, in some cases of congenital heart disease, emphysema, polycythemia, and abnormal fixation of hemoglobin with certain chemical compounds, there may be striking cyanosis with relatively little shortness of breath.

Common causes of dyspnea are:

1. Exertion. Age, sex, weight, general muscular tone, and the type and degree of activity to which the individual is accustomed are factors which must be kept in mind in evaluating the importance of dyspnea provoked by exertion It is

obvious, for example, that an obese, old person will experience marked difficulty in breathing after an amount of exertion that will hardly quicken the respiration of a trained athlete. *The relative ease with which dyspnea overtakes the normal person unaccustomed to exertion depends not so much on the condition of his heart and lungs as on the ability of the peripheral circulatory apparatus to supply by vasodilatation increased flow of blood to the muscles in use and by vasoconstriction to reduce blood flow to inactive parts, and on the capacity of his skeletal musculature to transform energy at the required rate* This serves to explain why a person accustomed to a particular activity will, in its performance, experience no breathing difficulty, whereas activity requiring no more energy but to which he is not accustomed will make him short of breath. *Altitude* must also be taken into account. In a rarified atmosphere one accustomed to living at a low altitude will become dyspneic with relatively little exertion; the permanent resident is physiologically adjusted by having high hemoglobin and red cell levels.

2 Obesity

3 Pain associated with respiratory movement

4. Heart disease

5. Pulmonary embolism.

6. Disease of the respiratory system, such as laryngeal, tracheal or bronchial obstruction, asthma, emphysema, tuberculosis, pneumonia, malignant disease, pleural effusion, and pneumothorax

7 Irritant gases

8 Anemia.

9 Debility or weakness, with or without anemia

10 Hyperthyroidism, with or without cardiac failure.

11 Marked distention of the abdomen as in ascites, tympanites, tumor or pregnancy.

12. Paresis of some of the respiratory muscles, as in poliomyelitis or progressive bulbar paralysis.

13 Neurocirculatory asthenia

In cases of dyspnea associated with disease of the respiratory tract, it is of some value to note whether the inspiratory or expiratory phase is chiefly affected As a rule, interference with flow of air in or above the larynx will produce inspiratory dyspnea, trouble below the larynx, such as bronchial asthma or emphysema, impedes expiration. In dyspnea due to most other causes, both phases are affected

Orthopnea. Dyspnea is so severe in recumbency that the patient is forced to remain upright to facilitate breathing and reduce distress

Polypnea. The rate of breathing is increased This almost always accompanies dyspnea and often, but not necessarily, hyperpnea

Hyperpnea. Respiratory movements are increased in depth but distress is minimal. The rate may be increased (*Kussmaul breathing*), normal, or diminished. Hyperpnea and polypnea are often entirely objective; the patient may be

unaware of any change in breathing. Barring exertion, one or both is most likely to be encountered in·

1. Diabetic coma.
2. Uremia
3. Certain acute infectious diseases, especially pneumonia.
4. General peritonitis.
5. Severe hemorrhage.
6 Fright, hysteria and other disturbed emotional states.

Bradypnea. Usually deep but occasionally shallow, slow breathing is most likely to occur in uremic or diabetic coma, drug poisoning, acute alcoholism, or intracranial disease

Asthmatic Breathing. In asthma, emphysema, severe bronchitis and sometimes in obstruction below the larynx, the expiratory phase of breathing is much longer than the inspiratory. Inspiration may become a short gasp, while expiration is prolonged to a drawn-out wheeze. Distress is pronounced

Cardiac Asthma. This term refers to paroxysmal dyspnea of the asthmatic type caused by pulmonary congestion and occurring in heart disease, especially left ventricular failure and tight mitral stenosis. It is featured by paroxysms of orthopnea and dyspnea, sometimes with long wheezing expiration, usually starting during sleep, exertion or an emotional upset and lasting from a few minutes to several hours It is often a bad prognostic sign

Cheyne-Stokes Breathing. This is an anomaly of respiratory rhythm in which short paroxysms of deep, rapid breathing alternate with periods in which no [illegible]

of respiratory movements to severe paroxysms of pronounced dyspnea and hyperpnea alternating with periods of apnea, lasting sometimes as long as 30 seconds A complete cycle may vary in length from 30–70 seconds When the breathing phase begins, the individual respirations are small and slow but gradually increase to a maximum in depth and rate, then gradually diminish until the point of apnea is reached The patient may sleep during the apneic periods and become restless, cough or awaken during the dyspneic phases

Cheyne-Stokes breathing, except for a mild form seen in normal persons during sleep and at high altitudes, occurs most commonly in advanced cardiac and renal disease, asthma, severe pneumonia, increased intracranial pressure and drug poisoning In disease, unless it is due to sedation, which it sometimes follows, it is usually a serious prognostic sign

Modified types of this phenomenon also occur. There may be rhythmic in-



Fig 8 6. Cheyne-Stokes respiration

crease and decrease in the depth and rapidity of respiration, without intervening periods of apnea. Or the onsets of hyperpneic and apneic phases may occur abruptly (*Biot's breathing*). The latter is often seen in meningitis

Restricted Breathing. Pain arising in the pleura or a musculoskeletal structure of the thorax may cut inspiratory excursion so short as to make breathing shallow, rapid and jerky This picture is seen in acute pleuritis, malignant disease of the pleura, fractured rib, separated costal cartilage, severe muscular or ligamentous strain of the thorax or upper back, and disease of the cervicothoracic spine. Rapid, shallow breathing can also be caused by abdominal distention.

Shallow Breathing. In peripheral circulatory failure, meningitis, and unconsciousness from various causes respirations are shallow and irregular. Phases of deeper breathing may intervene

Sighing Respiration. Normal rhythm is interrupted from time to time by a long, deep respiration Often a patient will give a history of being short of breath but careful questioning will bring out that he is actually referring to this occasional compulsive phenomenon, not to true dyspnea Most commonly found in nervous, fatigued or excitable persons, this variant is valuable in differentiating heart disease from some nervous states, especially neurocirculatory asthenia It is not a sign of cardiac or pulmonary disease, but may occur in peripheral circulatory failure

Stridulous Breathing. When there is obstruction to entrance of air at or near the glottis, a high-pitched, crowing or barking sound is heard during inspiration. This occurs in inflammation or edema of the glottis, laryngismus stridulus, laryngeal diphtheria, postpharyngeal abscess, laryngeal tumor or foreign body, and tracheal obstruction by foreign body, tumor or pressure from without. In pertussis, the whoop is a stridulous inspiration If intake of air is sufficiently hampered, the intercostal spaces and upper abdominal wall will be drawn inward during inspiration

Stertorous Breathing. The normal sound of respiration is obscured by rattling or bubbling due to vibrations set up within the upper respiratory tract or to secretions in the trachea or large bronchi It occurs in severe illness, especially moribund states, and is a bad sign The death rattle is an extreme form of stertorous breathing

Sternomastoid Breathing. In moribund patients, respiration is often irregular, gasping, and likely to be accompanied by a peculiar nodding movement of the head, the chin being thrown quickly upward during inspiration and falling slowly during expiration.

DIAPHRAGMATIC ACTION

Litten's Phenomenon. This term applies to the movements of the diaphragm as seen by the following procedure The patient lies on his back, chest bared, arms out and feet pointing toward a strong light Cross lights must be excluded. The observer sits at the patient's side, back toward the light. As the ribs rise with inspiration, a short, narrow shadow may be observed moving down the

axilla from about the seventh to the ninth or tenth rib. During expiration it returns to the starting point but is less readily seen Normally the excursion is about 6 cm., with forced breathing, 9 cm. The shadow is best visible in spare, muscular young men but can be seen in most healthy persons except the obese, or when respirations are shallow.

The mechanism is best understood by imagining a coronal section of the thoracic wall as seen from the front or back (Fig. 8 7). At the end of expiration, the diaphragm lies flat against the wall from its attachment up to about the sixth rib. During inspiration it moves away as it descends, thus allowing the expanding lung to enter the space between diaphragm and thoracic wall. This produces the shadow.

In basal consolidation, pleural effusion and emphysema, the shadow is absent on the affected side because the diaphragm is held away from the thoracic wall. Plueritic adhesions, obstructive atelectasis, and subphrenic abscess obliterate or reduce it by restricting phrenic motion. It is not ordinarily affected by abscess or tumor within the liver except when the lesion is exceptionally large. Hence the Litten method may be helpful in differentiating between intrathoracic disease and subphrenic abscess on the one hand, and enlarged liver on the other. X-ray studies are more valuable.

Restricted Movement. Obesity, greatly enlarged liver, and enlargement or distention of the abdomen by pregnancy, gas, tumor or fluid raise the diaphragm and interfere with free respiratory excursion Dyspnea may be produced, the occurrence of such complications as obstructive atelectasis and bronchopneumonia is encouraged

Disease of the lung or pleura, such as pneumonia, atelectasis, fibrosis, pleural effusion, and pneumothorax tend to interfere with phrenic movement on the homolateral side. The nature of the process will determine whether the diaphragm is elevated or depressed.

Injury to the phrenic nerve causes homolateral paralysis and elevation of the diaphragm. In bulbar palsy and bulbar poliomyelitis, bilateral paralysis is a serious and often fatal complication

In visceroptosis and hypertrophic emphysema, the diaphragm is low and its excursion limited

By causing alternating pressure changes within the abdomen and thorax,

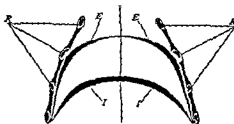


FIG 8 7 Excursion of diaphragm during forced respiration R, ribs, E, position of diaphragm at end of expiration; I, at end of inspiration

diaphragmatic activity is one of the factors promoting return flow of blood from the splanchnic area to the heart; when movement is diminished this mechanism is no longer fully operative.

THE PERIPHERAL VESSELS

The peripheral vessels in the various parts of the body are discussed in the pertinent chapters. In this section are included only those features which are more closely associated with disturbances of the circulation as a whole.

The Arteries

Pulsation. Pulsation of the carotids is seen in many normal persons, particularly those who are thin or nervous. Throbbing, noticeable particularly above the clavicles, is seen in heart disease, especially free aortic regurgitation, hypertension, *arteriosclerosis, severe anemia, coarctation of the aorta, and overaction* of the heart, as from excitement or thyrotoxicosis Visible pulsation in the subclavian, axillary, brachial and radial arteries, as well as in the large arteries of the lower extremities, may be produced by some of these causes

Excursion. Tortuous arteries in the extremities, especially the brachials, sometimes show lateral excursion, more rarely excursion upward and downward, synchronous with heart beat This occurs most commonly in spare, elderly men with extensive arteriosclerosis, and in free aortic regurgitation.

The Veins

The systemic veins provide a significant index of the state of the circulation. Pressure in the peripheral veins may be measured directly as described below, but information can also be obtained by careful inspection

Veins lying below the level of the right auricle have greater pressure and are *more full than those above it For example, when the arm hangs loosely at the* side, the veins are full, when it is held at the level of the auricle, they are flat If there is increased venous pressure, the arm must be raised to a higher level before emptying takes place The normal person, standing or sitting, *will show* distention of the veins of the lower extremities, but when he lies flat, the fulness will disappear

Distention. Inability of the heart to transfer sufficient blood from the venous to the arterial side causes backing up in the venous system, with resultant increased venous pressure and distention of peripheral veins This may be detected by inspection of the cervical veins

One determines the upper limit of distention of the external jugular vein and measures the distance between that point and the level of the manubrium. The significant measurement is not the height of the engorgement measured anatomically along the course of the vein but the *actual perpendicular distance* between the two levels. Engorgement secondary to external pressure such as that from a tight collar or tense muscles must be excluded

The cervical veins of a normal person in the upright position are collapsed *If he is placed in a* reclining position with his head resting on a pillow, the ex-

ternal jugular veins will probably be swollen in their lower parts, but the swelling will end at a point approximately level with the manubrium. Lowering the head will cause the swelling to extend upward in the vein, but the level, with respect to the sternum, will remain the same until the head is dropped below the plane of the sternum, whereupon the vein will become distended throughout. If, on the other hand, the head is passively raised, the distal level of distention will recede toward the sternum *When venous pressure is increased by local obstruction or cardiac failure, the swelling of the vein will extend to a higher level and in severe cases will be present even with the subject upright.* Occasionally the jugular vein does not remain superficial but runs more deeply in the neck in the upper part of its course, thus concealing distention which reaches a level actually higher than is apparent to the eye. Such an anomaly can be discovered by exerting digital pressure on the lower part of the vein and noting how far towards the jaw its course can be followed.

Pulsation Pulsation of the cervical veins is caused by their alternate filling and emptying, brought about by auricular action In contrast to the sharp pulsation of the carotid arteries, venous pulsation is undulating and rarely palpable It varies with respiration because of the changes in venous pressure secondary to changes in intrathoracic pressure It is best seen at the point of highest filling of the vein and, like venous filling, is related to body position In a normal person, venous pulsation is absent when he is upright, but as his head is lowered and the vein fills, it appears above the clavicle and moves upward toward the jaw as he approaches the supine position When venous pressure is elevated and the cervical veins are consequently distended with the patient upright, pulsation will be high in the neck, as pressure is further increased by lowering the head, the level of distention will extend further upward along the vein to a point where pulsation will disappear. Sharp systolic pulsation of



FIG 8.8 Distention of cervical veins due to congestive heart failure Patient is upright.

internal jugular veins, appearing deeper in the neck and reflected as outward thrusts of the overlying sternomastoid muscles, is observed in tricuspid regurgitation, structural or relative (*see* Chap 16)

Other superficial veins occasionally appear to pulsate, but usually this is transmitted pulsation from their accompanying arteries

Enlargement. Superficial veins of the neck, chest and abdomen may enlarge to form a collateral circulation as compensation for block in the normal venous pathways Examples are dilatation of the thoracic and abdominal veins in cirrhosis of the liver with obstruction to portal circulation and of upper thoracic and cervical veins due to interference of venous return by intrathoracic tumor. In contrast to venous distention associated with cardiac failure, dilatation secondary to obstruction is not accompanied by pulsation because of lack of free communication between the veins and the right auricle

Collapse. In peripheral circulatory failure, venous pressure is decreased by diminution of circulating blood volume The peripheral veins do not fill normally and appear collapsed. In severe cases, even if outflow of blood from a section of vein is prevented by digital or tourniquet pressure, the vessel distal to the point of block fills very slowly or not at all

Determination of Venous Pressure. When inspection of superficial veins is not satisfactory or when it is necessary to follow closely the progress of a case presenting chronic venous stasis, direct determination of venous pressure should be made The following procedure is used With the subject recumbent, his arm is placed on a support in such a way as to bring the antecubital space to the level of the right auricle Muscular relaxation is imperative Depending on the size of the thorax and thickness of the tissues of the back, the antecubital space will be at proper level if it is placed 10–12 cm above the table A needle of fairly large bore with a vertically attached graduated tube (Fig 8 9) in introduced into a large antecubital vein Clotting can be avoided by first rinsing the apparatus with physiological sterile saline or 2 per cent sodium citrate solution and performing the test quickly The height to which the blood rises in the tube represents the venous pressure in centimeters of blood (or water)

To minimize clotting risk, a more elaborate apparatus can be used A three-way stopcock is attached to the needle A syringe containing 2 per cent sodium citrate or physiologic salt solution is attached to a second outlet and a graduated glass tube to the third The latter must be vertical With the needle cut out by the stopcock, fluid is introduced from the syringe into the vertical tube in sufficient quantities to reach the anticipated level of venous pressure The valve is then turned to cut out the syringe and establish communication between needle and tube, the fluid in the latter will rise or fall to the level of venous pressure.

The normal venous pressure varies from 4–11 cm of water; usually it is 6–8 cm Prolonged or hard exercise may raise it to 10 cm. or more, probably because of increased flow of blood from capillaries into veins

Readings of 2–3 times the normal may be encountered in such disorders as

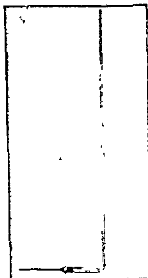


FIG 8 9

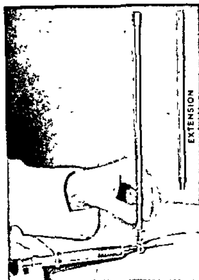


FIG 8 10

FIG 8 9 Simple apparatus for direct determination of venous pressure

FIG 8 10 More elaborate apparatus for direct determination of venous pressure (Courtesy Becton-Dickinson & Company)

moderate or marked congestive heart failure, venous obstruction due to thrombosis or compression, and constrictive pericarditis with inflow stasis. Increased venous pressure in congestive heart failure represents failure of the *right* side of the heart. Severe degrees of left ventricular failure, especially if acute, may exist without elevation of peripheral venous pressure

THE CAPILLARIES

Capillary Pulsation. The skin and mucous membranes show alternate flushing and blanching due to abnormal filling and emptying of the smaller vessels and usually associated with high pulse pressure This phenomenon can be seen by looking through a glass slide which is pressed gently against the skin of a fingertip or the vermilion border of a lip Sometimes gentle pressure on the fingernails will bring it out It is seen in cases of free aortic regurgitation and of large pulse pressure from other causes, such as thyrotoxicosis Since it also occurs in some normal individuals, and in any area where local hyperemia exists, its clinical significance is limited

THE SKIN

The skin of the thorax and back must be investigated for the indications of systemic disease, such as pallor, cyanosis, edema, jaundice, shininess, scars, and abnormal distribution and type of hair It is equally important to note the presence, distribution and character of any cutaneous lesions The eruptions of all the exanthematous diseases are usually found on the upper trunk. They may be

most prominent on the back, chest, or sides They are often confused with drug eruptions Important skin disorders which appear on the trunk are: acne vulgaris, dermatitis herpetiformis, scabies, urticaria, pediculosis vestimentorum, pediculosis pubis, pityriasis rosea, psoriasis, tinea versicolor, seborrheic dermatitis, impetigo contagiosa, ichthyosis, and pemphigus. The lesions of herpes zoster are particularly apt to occur on the thorax, almost always unilaterally. Since the eruption is usually preceded or accompanied by severe neuralgic pain, herpes zoster is often wrongly diagnosed as acute pleuritis especially if, to lessen discomfort, strapping or some counter-irritant is applied before the typical lesions appear.

9

PALPATION OF THORAX. PULSE. BLOOD PRESSURE

PALPATION OF THORAX

The Apex Impulse

Except in the presence of a thick chest wall or some pathologic disturbance such as emphysema or pleural or pericardial effusion, the heart beat can be felt over the lower precordium and the point of maximum impulse often can be more accurately determined than by inspection. For the region as a whole, the palm and fingers are used, for a more limited area, the fingertips. Pertinent facts regarding study of the impulse have already been described (*see* Chap 8). Different types of cardiac beat can be distinguished. the strong, forceful systole of a hypertrophied heart, the weaker, slapping beat of a failing heart, the sharp rap characteristic of advanced mitral stenosis, a slight extra apical thrust in gallop rhythm. Although palpation is often better than inspection for study of rate and rhythm, auscultation is even more reliable.

Percussion is also important in determining the size and position of the heart. If, for some reason, the impulse cannot be located and percussion is unsatisfactory, one must then resort to auscultation The apex can be assumed to be near the point at which heart sounds are most clearly heard, but this is only an approximation and not as accurate as one would like.

Thrill

Thrill is a series of vibrations transmitted through overlying structures to the palpating hand usually from the heart, aorta or thyroid It is comparable to, but less pronounced than the sensation imparted to the hand placed on the throat of a purring cat It can sometimes be more clearly distinguished by the palm, sometimes by the fingers Light palpation is usually more sensitive, but occasionally firmer pressure will reveal a thrill which has escaped gentler touch.

Cardiac Thrill. Cardiac thrill represents vibration of structures adjacent to a narrowed valve orifice, presumably set up by eddies in the blood current due to loss of streamlined flow. The three most important factors determining its intensity are speed of blood flow, degree of narrowing of the valvular orifice, and pressure differential between the two chambers The faster the flow, the narrower the orifice and the greater the pressure differential, the more intense the

thrill. The ease with which it can be felt is governed also by the thickness of chest wall and presence or absence of any intrathoracic abnormality which might interfere with transmission. In thin-chested persons and in those with overactive hearts, care must be taken to distinguish a true thrill from the sensation communicated to the palpating hand by the normal contraction wave A significant thrill is rarely palpable over the precordium except in the presence of a corresponding murmur.

SYSTOLIC THRILL This is almost always felt at the base, usually to the right of upper sternum, and often, by transmission, in the neck. It is most likely due to aortic stenosis, rarely to aneurysm. A systolic thrill best felt to the left of the sternum bespeaks congenital stenosis of the pulmonic valve, or ventricular septal defect At the apex, systolic thrill may represent extension downward of the thrill of aortic stenosis or, in rare instances, be caused by mitral regurgitation of high degree

DIASTOLIC THRILL Occurring almost always at the apex, this is usually due to mitral stenosis. It may occupy all or part of diastole If cardiac rhythm is regular, it is most intense just before systole (*presystolic thrill*), in the presence of associated auricular fibrillation it is felt in mid-diastole. Apical diastolic thrill may occasionally be transmitted upward toward the base.

Continuous Thrill. This is felt over the lesion. The most likely causes are:

1 Patent ductus arteriosus
2 Arteriovenous fistula
3. Hyperactive enlarged thyroid gland
4. Vascular malignant tumor

TACTILE FREMITUS

This term refers to tactile perception of the vibrations set up in the thoracic wall by phonation The observer places the palmar surfaces of the fingers or the medial edge of the hand and little finger on the subject's thorax and has him repeat a test phrase such as "one, two, three," or "ninety-nine" The fingers and hand should be relaxed and held against the body surface firmly but without undue pressure; strict uniformity of word formula and pitch and volume of tone must be maintained. Symmetrical areas of the thorax should always be compared in order to detect slight variations

Normally, fremitus increases in intensity in direct ratio to the depth, volume and resonance of the individual voice and is consequently greater in men than in women, and in adults than in children In women and children it may be too slight to be of any diagnostic value It is stronger in persons with thin-walled, flexible thoraces; it is perceived poorly or not at all in the obese or heavily muscled.

Fremitus also varies widely in different parts of the normal thorax In general it may be said that. (a) It is slightly greater throughout the right side than in corresponding areas of the left. (b) It is strongest over the upper thorax anteriorly and between and below the scapulae posteriorly. (c) It is poorly perceived over the scapulae and precordium

Increased Fremitus. Common causes are:

1. Pulmonary solidification, as in pneumonia, large pulmonary infarction, or extensive tuberculosis
2. Compressive atelectasis (some cases).
3. Pulmonary cavitation (some cases). Theoretically, fremitus should be absent or diminished over a cavity, actually, it is often increased because of the inflammatory density surrounding the cavity.

In any of these states, tactile fremitus is increased only if the bronchus leading to the affected part is open; if it is closed, fremitus is diminished or absent.

Decreased or Absent Fremitus. The usual causes are.

1. Pleural thickening, pleural effusion, and pneumothorax.
2. Asthma and emphysema
3. Obstruction of a bronchus

Theoretically, cavities not surrounded by a zone of infiltration give diminished or absent fremitus; actually they are seldom large enough to cause appreciable change.

FRICTION

Pleural Friction. Roughening of the pleural surfaces by an inflammatory process often produces a grating feeling or sound during respiratory movement, especially toward the end of inspiration It is best detected by auscultation, being palpable only if extensive. The most likely site is low or mid-axilla Occasionally, coarse, low-pitched rales will communicate a sensation which can be mistaken for pleural friction, auscultation will differentiate between the two.

Pericardial Friction. When the pericardial surfaces are inflamed, friction synchronous with heart beat may be heard and sometimes felt over the precordium If of slight degree it is most apt to be found near the fourth left costal cartilage Palpable friction serves only as a confirmatory sign; auscultation is more important

Joint Friction. Friction produced during respiration by movement of the shoulder joint or its related muscles and tendons occasionally can be felt as a grating sensation, or more likely heard as a rumbling, grating sound usually in the scapular region and extending upward toward the shoulder. Confusion of this variant with pleural friction can be avoided by having the patient assume a position in which all the muscles are relaxed, the joint or tendon friction will no longer be evident

TENDERNESS

Local tenderness is most likely due to fractured rib, separated costal cartilage, acute pleuritis, acute pericarditis or some inflammatory lesion of the thoracic wall Precordial tenderness, especially over the apex, is common in neurocirculatory asthenia and occasionally encountered in mitral disease.

PULSATION OF THORACIC WALL

Abnormal pulsations of the throacic wall may be palpable as well as visible (see Chap. 8)

The Skin

Undue roughness, smoothness, or dryness of the skin and the character of certain cutaneous lesions can often be better appreciated by palpation than by inspection

THE PULSE

Palpation of the arterial pulse remains an important clinical procedure despite the development of more exact clinical and laboratory methods of cardiovascular study It provides a quick estimate of the general condition of the patient and the state of his circulation, and affords reliable evidence of certain cardiac disorders, such as arrhythmias, pulsus alternans, and lesions of the aortic valve

The term, *the pulse*, in clinical usage, refers to the wave in the radial artery, which, because of its superficial position and easy accessibility, is the one ordinarily palpated The wave does not represent forward movement of a column of blood propelled through the vessels. It is actually an advancing wave, produced by sudden ejection, during cardiac systole, of blood into the aorta; it proceeds through the arterial system in advance of the blood ejected by systolic contraction and independent of the speed of blood flow. It may be compared to the wave set up by a stone thrown into a quiet pool

The radial pulse is usually found on the anterior surface of the wrist just medial to the styloid process of the radius and is best felt with the subject's wrist relaxed and slightly flexed Both radial pulses should be palpated simultaneously, at least at the first examination, and thereafter if any discrepancy is noted

Occasionally, one or both arteries follow an anomalous course, usually deeper in the arm or running superficially and more laterally along the lower part of the radius. Under such circumstances, an inexperienced observer may report in alarm that the patient is pulseless when, as a matter of fact, he has simply failed to locate the radial artery Another source of error is the unusually small artery. Here, hurried or inexpert palpation may lead to the incorrect conclusion that the pulse is weak This mistake can be avoided by determining the degree of digital pressure necessary to obliterate the pulse wave or by sphygmomanometric determinations of the blood pressure If the artery is merely small, the readings will be normal, if the pulse is actually weak, systolic or diastolic pressure, or both, will be low.

If the radial artery is inaccessible because of its anomalous position or some factor such as a surgical dressing, the temporal, facial, carotid, brachial or femoral artery may be substituted, and a notation made to this effect

If arterial disease or some such disturbance as aneurysm or coarctation of the aorta, which might affect the flow of blood to one or more extremities, is suspected, the character of the pulse in the brachial, axillary, popliteal and dorsalis pedis arteries should also be determined

In examining the pulse, the observer places his second and third fingers, never the thumb, over the artery, and notes the following.

1. Rate of pulse.

2. Rhythm of pulse.
3. Condition of arterial walls.
4. Compressibility and tension of pulse.
5. Size and shape of wave.

Rate of Pulse

There is no absolute normal. The rate varies in different individuals and in the same individual under different circumstances In the adult male at rest during the waking state, it ranges between 60 and 80 per minute, in females, between 70 and 90 During sleep it is slower, usually ranging between 50 and 70 in men and 60 and 75 in women, rates as low as 45 to 50 are not uncommon The rate increases on rising to a standing position, after eating, during exercise, in febrile states, and in a large number of diseases Emotional stress also quickens the rate; in high-strung persons acceleration during the examination may make it difficult to determine the basic range In a resting adult, a rate persistently over 100, if emotional factors are excluded, should be considered evidence of disease until proved otherwise In some families, a slow pulse (below 60) is a hereditary trait and does not indicate disease. Athletes in training often show resting pulse rates as low as 40 or 50. In children, the pulse averages about 130 at birth and remains above 100 until the third year

The various causes of alteration of the heart rate are too numerous to be enumerated.

The radial pulse may not provide a true indication of the heart rate, for, with certain disturbances of cardiac rhythm, such as premature beats or auricular fibrillation, a considerable number of beats may not be sufficiently strong to convey impulses to the peripheral vessels. The pulse rate at the wrist must always be checked by a count of the cardiac beats at the apex.

Rhythm of Pulse

Normally the pulse is regular in rhythm and force. However, an alternate quickening during inspiration and slowing during expiration (*sinus arrhythmia*) is normal in almost all children and adolescents, and in some adults It is more noticeable with a slow rate

Interruption of a regular rhythm by a momentary irregularity occurring occasionally or at frequent intervals is often encountered This is most commonly due to *premature contractions* A single premature contraction or the final one of a series is often followed by a pause, especially when the abnormal beats are of ventricular origin Similarly, a pause is noticeable if the premature contraction, although heard over the heart, lacks sufficient strength to produce a wave in the peripheral artery It is to this form of arrhythmia that the term "intermittent pulse" is most often applied, but since it is also used in cases of heart block with dropped beats, and even in auricular fibrillation, the term should be avoided At times premature beats may be so frequent that only one, two or three regular pulse beats occur between them. If single premature beats replace every other normal contraction it is obvious that the pauses which follow them

will cause a coupled rhythm at the apex. When these premature beats are strong enough to produce pulse waves at the wrist, the pulse will also be coupled (*pulsus bigeminus*); otherwise it will be halved. Premature beats tend to disappear when the heart rate is quickened.

Another but less common form of irregularity is that caused by *dropped beats* due to heart block. Here the rhythm is interrupted by a pause because the heart actually misses a stroke In contrast to its effect on premature beats, acceleration of heart rate *accentuates this irregularity by increasing the number of* dropped beats.

Absolute irregularity in rhythm and force of the beats is almost always due to *auricular fibrillation*. Except after administration of therapeutic doses of digitalis, exertion causes undue acceleration of rate and increases the difference in strength of individual beats, thus accentuating the irregularity. In contrast, the irregularity caused by premature beats usually disappears with exertion or other cause of increased rate. In auricular fibrillation, particularly if the rate is rapid, many beats lack sufficient force to produce a pulse wave at the wrist (*pulse deficit*) or even at points in the arterial circulation nearer the heart Beats which do appear at the wrist are not of equal strength In the presence of auricular fibrillation or premature beats the cardiac rate must be determined by examination of the heart, to rely solely on the pulse may lead to serious consequences.

Condition of Arterial Walls

If flow of blood through an artery is stopped by digital pressure, the normal artery below this point cannot be felt In arteriosclerosis, the vessel is palpable and, depending on the degree of pathologic change, varies from a soft tube just felt when the examining finger is rolled over its surface to one which is hard, rigid, and perhaps beaded by calcium deposits Since changes in pressure within a vessel are obviously more clearly reflected in one with elastic walls than in one which has become thickened and less elastic from arterial disease, the condition of the arterial walls must always be noted and taken into consideration when one is studying the character of the pulse The inelasticity of a rigid artery, often causes a strong pulse wave to appear weak

Compressibility and Tension of Pulse

A rough estimate of systolic pressure may be obtained by noting the degree of digital pressure necessary to obliterate the pulse wave *With the more proximal* of his two palpating fingers the examiner presses the artery against the underlying bone until the pulse wave is no longer felt with the distal finger The diastolic pressure can be roughly estimated by noting the degree of hardness (*high tension*) or softness (*low tension*) of the pulse between beats The pulse of low tension appears to collapse so that nothing is felt The pulse of high tension is perceptible between beats as a cord which can be rolled beneath the fingers. These distinctions are *obviously by no means as clear-cut in an* arteriosclerotic vessel. In every case, the observations should be checked by sphygmomanometric determinations.

SIZE AND SHAPE OF WAVE

Although sphygmographic tracings are the only exact means of determining the character of the pulse wave, it can often be estimated by the trained finger. The three most important factors influencing the size and shape of the pulse wave are: (1) the quantity and velocity of left ventricular discharge; (2) the peripheral resistance, (3) the elasticity of the arterial system. The pulse wave will be small, for example, when the artery is narrowed, when cardiac output is small because of ventricular weakness or diminished venous return, or when the speed of discharge is slow, as in aortic stenosis. It is also small when peripheral resistance is increased and arterial elasticity is diminished, as in many instances of long-standing arterial hypertension Conversely, the pulse wave will be large when cardiac stroke volume is increased, as in bradycardia, or when peripheral resistance is diminished, as in thyrotoxicosis and high fevers. These variations are also conditioned by the elasticity of the arterial walls; they will not be as apparent in an artery which is rigid In fact, it is at times impossible to distinguish between a normal pulse in an artery stiffened by arterial disease and a high-tension pulse in a relatively normal artery.

Normal Pulse. From what has already been said, it should be apparent that there is considerable variation in the character of the pulse, even in normal persons What constitutes a normal pulse and what is abnormal can be learned only by long experience. To the trained finger the normal wave appears to rise fairly sharply but not abruptly, is sustained for a moment, and then disappears Following exertion or when the subject is nervous, it may be more forceful and rise somewhat more sharply, it may show a quicker and stronger impact against the examining finger and seem to disappear more quickly.

Bounding Pulse. Sometimes called *collapsing pulse*, this variation is best detected with the arm held aloft The wave swings sharply upward, usually reaches a level higher than normal, and disappears quickly It is due to a shortened ventricular systole and concomitant diminution of peripheral resistance. Bounding pulse is found is severe febrile states, thyrotoxicosis, emotional disturbances, and other disorders causing peripheral vasodilatation The low tension during diastole makes the systolic wave appear strong, conveying the impression that the pulse is good, when actually it may be poor. An extreme form of bounding pulse is the *Corrigan pulse*, seen in free aortic regurgitation, arteriovenous fistula and pronounced patent ductus arteriosus

Plateau Pulse. In contrast to collapsing pulse, the wave rises gradually, is sustained for a longer period, and falls gradually This occurs in aortic stenosis: blood is discharged slowly into the arterial system through the narrowed aortic ring

Thready Pulse. The rate is rapid, the wave appears quickly, is small and disappears quickly, giving the impression of very little strength. This is found in some cases of severe myocardial failure and in peripheral circulatory collapse

Dicrotic Pulse. The descending limb of the normal arterial pulse tracing



FIG. 9.1. Sphygmographic tracing of normal pulse

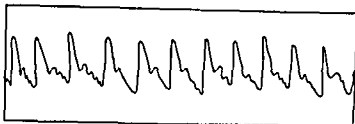


FIG. 9.2 Sphygmographic tracing of bounding pulse



FIG 9.3. Sphygmographic tracing of plateau pulse.

shows a brief interruption indicated by a notch, which is followed by a small secondary wave. This usually cannot be detected by palpation Under certain circumstances, especially in fevers, the notch may be deep and the amplitude of the secondary wave sufficiently increased to be palpable in the radial pulse (*dicrotic wave*) When the heart rate is rapid, the secondary wave may be felt *just prior to the following primary wave Plateau pulse may show an* extra wave on its ascending limb (*anacrotic wave*) Neither of these variations has any special clinical significance

Pulsus Alternans Rhythm is normal, but a strong beat and a weak beat occur alternately. This may be noted by careful palpation; lesser degrees are detected only with the sphygmomanometer or sphygmograph *Pulsus alternans is an important and grave sign in cardiovascular disease;* it usually indicates serious *left ventricular weakness It must always be differentiated from pulsus bigeminus* due to premature beats In the former the beats are equally spaced, in the latter the strong beat and the following weak beat are coupled

Pulsus Paradoxus. In normal persons, a slight, gradual change in the amplitude of arterial pulse wave can often be observed through the respiratory cycle: the wave is highest at the peak of inspiration and lowest at the end of expiration. In well-marked thoracic-type respiration, the opposite may be true, the wave *being greatest at the end of expiration (pulsus paradoxus).* Since the normal variation is only 2–3 mm Hg, it usually can be detected only with the sphygmomanometer. Pulsus paradoxus of this degree is ordinarily of no clini-

cal importance. *A more extreme form often readily detected by palpation is found in cardiac tamponade, which is usually seen in rapidly developing pericardial effusion, and in constrictive pericarditis.* It sometimes occurs in peripheral circulatory failure, severe emphysema and asthmatic bronchitis.

ARTERIAL BLOOD PRESSURE

Method of Determination

The term blood pressure always refers to the systemic arterial blood pressure, unless otherwise specified Every physical examination should include sphygmomanometric determinations preferably made while the cardiovascular system is being examined. Readings taken shortly after exertion or when the subject is tense or emotionally disturbed are not dependable. In such instances basic systolic and diastolic levels must be established by taking additional readings toward the end of the interview and at subsequent visits

Because the blood pressure varies in vessels of different caliber it has become standard clinical practice to use the brachial artery. Reports always refer to brachial observations unless otherwise noted. The record should indicate which arm was used and whether the patient was standing, sitting or recumbent. Since, under certain circumstances (such as atrophy of the arm or obstruction to blood flow in the innominate or a subclavian artery) the pressure may differ in the two arms, one should always take readings on both arms at the first examination and subsequently if an any appreciable difference is found If for some reason the arms cannot be used, arterial pressure can be taken in the femoral arteries, where it is normally 20 to 40 mm higher than in the brachials Femoral artery readings should also be taken for comparison with brachial readings whenever interference with arterial flow by some lesion such as tumor, aneurysm, or congenital coarctation of the aorta is suspected.

The sphygmomanometer commonly employed consists of a cloth-encased, inflatable rubber cuff attached by rubber tubes to a mercury or aneroid manometer and a bulb air-pump Air forced from the pump is distributed simultaneously into the cuff and the manometer, the pressure in the two parts is the same An adjustable valve between the pump and the cuff permits the observer to release air and thus reduce pressure in the system at any desired speed. The mercury-type machine is somewhat more accurate than the aneroid, the latter, however, is satisfactory for ordinary purposes, provided it is standardized from time to time Readings are registered in millimeters of mercury.

Measurement of Systolic Pressure by Palpation. For this method, as well as those described below, the patient should be comfortably seated or recumbent with the arm relaxed at his side, supported at heart level and the forearm slightly flexed and pronated If the arm is in other positions, especially outward rotation or abduction, the brachial pulse may be diminished or obliterated. One must be certain that the limb is not constricted by a tight sleeve or other hindrance to free flow of blood The cuff, completely deflated, is snugly fitted around the upper arm, with its inflatable portion over the inner aspect and its

lower border a few centimeters above the antecubital space. Accurate results cannot be obtained with a cuff which bulges or becomes displaced with inflation

With the fingers of one hand on the patient's radial artery, the observer pumps air into the cuff until the radial pulse has been obliterated. By slightly opening the exhaust valve, air is gradually permitted to escape until the wave is just felt; the reading at this point indicates *the systolic pressure. Rapid release* of air must be avoided; otherwise the manometer readings will lag behind the decline of pressure in the cuff. To avoid venous congestion, observations *should be made with* dispatch. When successive readings are being taken, the cuff must be completely deflated for several minutes between observations.

The pressure in the inflated cuff must of necessity overcome not only the pressure of the blood within the artery but also the resistance of the arterial wall itself and of the surrounding soft parts. This tissue resistance is ordinarily of no clinical importance. When the artery is extremely arteriosclerotic, however, its incompressibility hampers both palpatory and auscultatory determinations.

The palpatory method is ordinarily not as accurate as the auscultatory method but should always be employed to avoid errors caused by the "auscultatory gap", described below.

Measurement of Systolic Pressure by Auscultation. The receiver of the stethoscope is placed over the brachial artery just peripheral to but not touching the cuff; it is held firmly against the skin but without undue pressure The pulse is first obliterated by inflating the cuff Air is now gradually released, and a series of tapping sounds in rhythm with the heart beat will become audible as blood begins to reappear in the artery below the cuff The point at which these sounds are first heard indicates the systolic pressure.

Failure to recognize the presence of an auscultatory gap is a frequent source of error encountered especially in cases of hypertension. This gap is a zone of silence appearing 10–30 mm. below the true level of systolic pressure and continuing for 20–40 mm downward toward the diastolic level Should it be present, it is obvious that the observer, in attempting to obtain the systolic pressure by the auscultatory method, might first listen in the zone of silence and consider the sound appearing at its lower level as the true systolic reading; actually he will have missed sounds which he would have heard had he originally inflated the cuff to a higher pressure If, for example, a patient has a systolic pressure of 250 mm Hg, he might show an auscultatory gap with no sounds audible while the pressure in the cuff is in the 230–200 mm. range. An inexperienced observer might at the start raise the pressure in his machine to 220 mm. and, hearing no sound, gradually let out the air until he heard sounds coming through at 200 mm He would then wrongly assume this to be the systolic blood pressure, having entirely missed the sounds coming through in the 250–230 zone Had he first used the palaptory method, he would have avoided the error, since this would already have shown the systolic pressure to be well over 200 mm.

Measurement of Diastolic Pressure. Except in recording instruments only



FIG 9 4 Auscultatory determination of blood pressure with aneroid sphgmomanometer

the auscultatory method is employed for determination of diastolic pressure. If pressure in the machine is gradually reduced by release of air, the systolic sounds audible at the level of systolic pressure will become louder and, although changing somewhat in character, remain fairly constant until a point is reached 20 mm or more below the systolic reading Here the relatively sharp tapping sounds change to dull, feeble thuds, the reading at this point of change represents the diastolic pressure In most persons, the arterial sound will cease entirely at a point 5 to 10 mm lower, but *diastolic pressure is indicated at the point where the change occurs in the character of the sounds, not at the point of their disappearance* If there is a difference of more than 10 mm. between the two points, both readings should be recorded. In cases of pronounced aortic regurgitation or peripheral vasodilation, a sound synchronous with pulse beat [illegible]

which it rarely is.

Measurement of Femoral Arterial Pressure. The technique is similar to that just described. The patient should be recumbent A larger cuff is preferable; this is fitted around the thigh with its lower edge a few centimeters above the popliteal space One palpates and listens over the popliteal artery.

Normal Blood Pressure

In normal healthy adults, *the range of systolic blood pressure is 90–120 mm Hg; of diastolic pressure, 60–80 mm.* As a general rule when systolic pressure tends to be in the lower range, the diastolic follows suit, and *vice versa* Before middle life, the systolic pressure in women tends to average 5–10 mm. lower than in men of corresponding ages. Beyond middle life there is no appreciable difference In children, the figures are lower than in adults, averaging 50–60 mm. systolic and 30–40 mm. diastolic at birth and gradually increasing to adolescence when there is a somewhat sharper rise to the adult level *Pulse pressure,* the difference between the systolic and diastolic pressures, usually ranges between 30 and 40 mm Hg.

The blood pressure in any normal person is subject to considerable variation; the systolic more so than the diastolic. Both systolic and diastolic pressures are usually higher in the upright than in the recumbent position, in asthenic persons or those with habitually low blood pressure, the systolic may be lower on standing During sleep systolic pressure is normally lower than in the waking state, sometimes as much as 25–30 mm Changes also occur normally *in response to such everyday factors as exertion, emotion, stress, and pain* These variations are more pronounced in subjects whose pressures tend toward the lower limit of the normal range than in those with the higher level The diastolic pressure should receive as much if not more attention than the systolic pressure because it is less affected by these factors, although this advantage is somewhat offset by the fact that the diastolic pressure cannot be measured as accurately as the systolic The diastolic pressure is especially important because it represents the constant load imposed on the walls of the arterial system and more accurately reflects the degree of peripheral resistance A high diastolic is often of greater significance that a high systolic Conversely, a low systolic has more significance than a low diastolic since the former reflects the strength and stroke-volume of the left ventricle

It must be emphasized that because of the fluctuations in blood pressure brought about by such factors as those mentioned above, a single reading is rarely sufficient. Observations taken on several different days, perhaps at weekly or monthly intervals and as nearly as possible under basal conditions, are necessary before one can definitely state that a person's blood pressure falls inside or outside the normal range.

High Blood Pressure

It is impossible to draw a sharp line between normal and high blood pressure As a general working rule, systolic pressure over 150 mm or diastolic over 90 mm persisting before middle age should be regarded as abnormal if such factors as exertion and emotional stress are excluded Beyond midlife the upper limit of normal may be somewhat higher *In any event, a systolic or diastolic pressure which tends to climb through the years must be viewed with concern.*

Pathologic high blood pressure is caused by the disorders listed below. The first five account for all but a small number of cases.

1. Essential hypertension, also known as primary, vascular or arterial hypertension, or hyperpiesia.

2. Nephritis, pyelonephritis, chronic destructive renal lesions, such as hydronephrosis or congenital cystic kidneys, and systemic diseases involving the kidneys, such as amyloidosis and periarteritis nodosa.

3. Interference with urinary outflow, as by prostatic obstruction, ureteral stricture, and impacted calculus, or by "spinal cord" bladder.

4. Aortic valve insufficiency, syphilitic or rheumatic. The diastolic pressure is below normal.

5. Thyrotoxicosis. Systolic pressure is moderately elevated; the diastolic is usually lowered.

6. Increased intracranial pressure, as in brain tumor or intracranial hemorrhage.

7. Toxemia of pregnancy.

8. Complete auriculoventricular heart block. The systolic pressure is high, the diastolic is normal or slightly lowered.

9. Adrenal tumor, cortical or medullary.

10. Obstruction to blood flow through the aorta by such causes as coarctation or pressure from aneurysm or tumor. Pressure is elevated in the arteries proximal to the obstruction and normal or lowered distally.

11. Pituitary basophilism.

12. Polycythemia vera (sometimes).

Low Blood Pressure

The statement above concerning the line of demarcation between normal and high blood pressure pertains also to low blood pressure. In general, hypotension is indicated by systolic pressure persistently below 90 mm. and diastolic below 40–50 mm. Many normal adults have a systolic pressure of 90–100 mm., a diastolic of 50–60 mm. This may be a familial tendency and is also likely to be encountered in asthenics and visceroptotics. Such persons are often unnecessarily alarmed by being told that they have low blood pressure. Actually these levels or those even lower are by no means incompatible with good health. In fact, many observers are of the opinion that persons whose levels fall within these ranges are less likely than others to become hypertensive in later years.

Many persons whose pressures remain in the lower ranges of normal complain of such varied symptoms as easy fatiguability, dizziness, susceptibility to cold, mental depression, backache and insomnia. These patients are often classified as cases of essential hypotension. It is our belief, however, that the relatively low pressure is not the cause of the symptoms, but is rather one of the manifestations of a clinical syndrome the exact cause of which is not understood.

Hypotension may be relative rather than absolute. A patient may show blood

pressure readings within normal limits; yet in his case they may actually indicate hypotension since they are lower than the level previously maintained. Since fall of blood pressure is frequently associated with cardiac failure, the presence of a normal or low blood pressure in a case of cardiac failure does not exclude hypertension as a possible cause of the trouble.

Acute Hypotension. This is seen most commonly in:

1 Vasovagal syncope (fainting) from such causes as emotional disturbance or other form of stress.

2 Postural hypotension Blood pressure is normal when the patient is lying down but drops suddenly when he stands, causing weakness, blurred vision, dizziness, and perhaps syncope. Additional manifestations are deficient sweating, local or general, and failure of the heart rate to accelerate when the patient stands The chief characteristic of this syndrome is inadequate vasoconstriction on the arterial side with resultant fall in pressure. A like picture lasting for weeks or months is apt to occur following dorsolumbar sympathectomy A somewhat similar syndrome is that described by MacLean and Allen[1] as orthostatic hypotension, due to inability of the venous side of the circulation to return sufficient blood to the heart The syndrome is essentially the same but the symptoms are often apt to occur in the morning when the patient arises and lessen during the day. In contrast to the postural hypotension just described, the low blood pressure is usually accompanied by acceleration of the heart rate In some cases with the same clinical signs, the heart rate accelerates on standing, but hypotension does not develop.

3. Stimulation of a hypersensitive carotid sinus.

4. Peripheral circulatory failure, as seen following serious injury or operation or in such disturbances as physical exhaustion, burns, hemorrhage, and myocardial infarction.

5. Acute cardiac failure, such as occurs with myocardial infarction, severe rheumatic myocarditis, or a prolonged attack of paroxysmal tachycardia. In congestive failure, the pressure may fall, but sometimes it rises until the terminal stage

6 Adams-Stokes syndrome in severe heart block.

7 Cardiac tamponade associated with rapid accumulation of pericardial fluid

8 Acute adrenal failure

9. Administration of vasodilator drugs such as nitroglycerine or amyl nitrite

Chronic Hypotension. The most likely causes are:

1. Any chronic wasting disease.

2. Addison's disease, or any form of chronic adrenal insufficiency.

3. Hypothyroidism

4 Severe mitral stenosis (sometimes). If present, the hypotension is mild

[1] MacLean, A R, and Allen, E V Orthostatic Hypotension and Orthostatic Tachycardia J.A M.A. 115. 2162 (Dec 1940)

Variations in Blood Pressure

Rhythmic Variations. The blood pressure in normal persons may be 2-3 mm. higher at the peak of inspiration than at the end of expiration. *Pulsus paradoxus,* in which the pressure rises at the end of expiration (see above), and *pulsus alternans* (see above), can often be detected with the sphygmomanometer when not of sufficient degree to be discovered on palpation of the pulse.

Irregular Readings. These are produced by variations in strength of heart beat, as in premature beats and auricular fibrillation When the pressure in the sphygmomanometer is raised to the level at which the strongest pulse waves come through and can thus be heard or felt distal to the cuff, the weaker waves are blocked by the pressure in the cuff and hence are not detectable beyond it. They may, however, appear when the pressure in the cuff is reduced to a lower level Some beats may be so weak as not to reach the periphery at all In auricular fibrillation, especially with rapid ventricular rate, there is little or no uniformity in strength of beats and accurate determination of blood pressure is impossible One can only estimate the diastolic and systolic pressures by noting the levels which obtain for the greatest number of beats The record should show that the readings are merely an estimate.

Discrepancy between Arms. A difference of 5-10 mm in the systolic and diastolic levels in the two arms is not infrequently found in normal persons. A greater discrepancy is usually due to one of the following causes:

1. Disparity in size of the arteries
2. Interference with the flow in the innominate or a subclavian artery by external pressure as from tumor, aneurysm or cervical rib
3. Blocking of the mouth of the innominate or a subclavian artery by syphilitic aortitis.
4. Thrombosis or embolism.

Raising the pressure in one extremity by exposing it to cold may bring about a temporary difference between the two sides

Abnormal Discrepancy between Upper and Lower Extremities. Higher pressure in the arms than in the legs—the reverse of normal—is brought about by interference with blood flow to the lower part of the body by such disturbances as:

1. Congenital coarctation of the aorta
2. Pressure on or obstruction of the aorta by saccular or dissecting aneurysm, tumor, or thrombus
3. Narrowing or obstruction of the iliac or femoral arteries

Pulse Pressure

Diminished Pulse Pressure. Pulse pressure slightly below the usual normal of 30-40 mm. is found during sleep and fatigued states More pronounced diminution is usually due to one of the disturbances producing acute hypotension as indicated above, pronounced aortic or mitral stenosis, or constrictive pericarditis

Increased Pulse Pressure. This is caused by:

1. Exercise, excitement, thyrotoxicosis, fever and other causes of overactive heart with or without peripheral vasodilatation

2. Hypertension. The systolic and diastolic pressures are both high but the systolic rise is proportionately greater

3 Free aortic regurgitation, arteriovenous fistula, or patent ductus arteriosus of more than slight degree Systolic pressure is high, diastolic low

4 Complete heart block Systolic pressure is high, diastolic, normal or low

PERCUSSION OF THORAX

TECHNIQUE

Percussion is the method of examination based on interpretation of the sounds and the sense of resistance encountered upon striking the chest a series of strokes. It has to some extent been neglected since the development of roentgenology but still retains much of its importance. No other method of physical examination requires so much practice and is so seldom mastered. The greatest difficulty arises from the necessity of being simultaneously active and passive, the one who percusses and the one who listens Successful percussion requires that the strokes be delivered automatically and the whole attention concentrated on listening to the sounds or feeling the resistance.

Since the chief end of this method is the correct interpretation of the sounds elicited, it is helpful but not necessary to have a musical ear. Judgment can be formed as accurately on the quality or intensity of the note as on its pitch. The sense of resistance is a further and at times more valuable source of information

Auditory Percussion

Percussion, in this country, is usually performed with the fingers alone; in some foreign countries instruments are employed There are two methods, the *direct* and the *indirect*, the latter being more commonly used.

The direct method refers to the application of strokes directly to the body surface by one or more fingers. Ordinarily, the pads, not the tips, are employed.

The indirect method is that of striking blows not directly upon the body but upon an intermediate object placed on its surface It is customary to strike with the right middle finger (*plexor*), and to use the left middle finger as the intermediate object (*pleximeter*)

The patient should be sitting or standing in a position which excludes all tilting or twisting and in which the two sides of his body are as nearly symmetrical as possible The muscles must be completely relaxed and the patient instructed not to hold his body rigidly nor expand his thorax When the back is being examined, the head should be bent downward, the shoulders slack and drooping, and the back bent slightly forward in a relaxed attitude If the patient is seated, the arms should rest loosely on the thighs or be folded across the lap During examination of the axillae, the hands are best placed on the hips with the elbows out In the scapular region better results can be obtained

if the scapulae are drawn laterally by bending the back far forward and crossing the arms over the chest, placing the hands on the shoulders. The bed patient who cannot sit up must be turned from side to side for percussion of the back, a procedure which is not altogether satisfactory because homologous areas cannot be readily compared.

The usual method of percussing the lungs is to start at the top and proceed *downward, always* comparing corresponding areas. When determining the border of an organ it is best to percuss successive points along a line running at right angles toward it, observing when a change of note occurs

The following rules must be observed·

1. Press firmly on the body surface with the pad or tip of the left middle finger. Raise the rest of the middle finger as well as the others away from the surface in order to avoid interference with vibrations. The amount of pressure required varies with the thickness of the body wall at the point of percussion; the thicker the chest wall, the greater the pressure necessary to deaden the vibrations of the structures overlying the lung.



Fig 10 1



Fig 10 2

Fig 10 1 Correct method of indirect percussion Only the pad of middle finger of left hand is in contact with body wall Right hand moves from wrist (Minimal changes in percussion note can sometimes be better detected with tip of plexımeter finger in contact with the wall) Note related position of subject for percussion of back

Fig 10 2 *Incorrect* position of left hand Thumb and fingers all in contact with body wall



Fig 10 3 Fig 10 4

Fig 10 3 Incorrect use of right hand, which is shown in two positions to demonstrate striking of blow from elbow instead of wrist Left hand also incorrectly placed

Fig 10 4 Correct position of patient for percussion of interscapular region

2. Deliver a quick, perpendicular, rebounding stroke with the pad of the right middle finger upon the left middle finger over the second phalanx, imitating as far as possible with the right hand the action of a piano-hammer The quicker the percussing finger is withdrawn after striking, the clearer will be the note obtained. (Left-handed percussors should keep the finger of the right hand on the chest and strike with the finger of the left hand)

3 Strike from the wrist and not from the elbow (Figs. 10 1 and 10 3) The wrist must be completely relaxed. In very light percussion, sufficient force may be obtained by using only the finger

4 Keep the percussing finger bent at right angles as in Fig 10 1

5 Let all the strokes on any one part of the chest be uniform in force. The force with which they should be struck depends upon the purpose for which the percussion is being used—that is, what organ we are percussing—and also upon the thickness of the body wall For example, it is necessary to percuss very strongly when examining the back of a muscular man, where an inch or two of muscle intervenes between the pleximeter and the lung Over the chest in the axillae and the lower thorax posteriorly the muscular covering is thinner, hence a lighter stroke suffices In children, the emaciated, or any person whose muscular development is slight, percussion should be just sufficient to elicit clear sounds Although sometimes necessary, heavy percussion is never entirely

satisfactory, because each sound elicited comes from a relatively large area of the thorax and consequently does not provide information concerning a sharply localized area If a carpenter tapping a wall to locate a stud strikes too hard he will fail to find it, because the blow delivered on a spot behind which the beam is situated is so forcible that it will bring out the resonance of the neighboring hollow parts. The same applies to medical percussion

The best percussion is that which is just forcible enough to elicit a clear faint sound over air-containing tissues (lung, stomach, or intestine), without setting surrounding structures in vibration. Over solid organs a percussion blow of the right strength should bring out a faint, deadened sound.

Palpatory Percussion

In auditory percussion the main object of study is the sound elicited; the resistance encountered by the fingers is noted incidentally In palpatory per-



Fig. 10 5 Direct percussion One or all fingers may be used. This is also the best method for palpatory percussion.

cussion, attention is focused primarily on the amount of resistance encountered and the character of the vibrations set up by the stroke. Most observers prefer the direct method; some, the indirect. Both are useful and should be tried when the findings are obscure. Indirect palpatory percussion is similar in technique to that of indirect auditory percussion except that the strokes are lighter. In the direct method one taps the body wall lightly with the pads of one or more fingers, being sure to deliver quick, bounding strokes with prompt withdrawal of the percussing fingers.

Palpatory percussion is particularly valuable in detecting fluid or solid tumor in the thorax and at times in determining the borders of the heart or other organs.

CONDITIONS HAMPERING EFFECTIVE PERCUSSION

1. Abnormal thickness of the body wall, such as obesity, increased musculature, large breasts, and edema.

2. A capacious bony thorax, causing excessive vibrations with resultant difficulty in distinguishing between various shades of resonance.

3. Distention of stomach or bowel by gas, causing tympany on percussion of the lower left chest anteriorly or laterally, or reducing normal liver flatness.

4. Incorrect position of subject. For example, corresponding areas in the two lungs cannot be compared if the subject is twisted, if the muscles on one side are more taut than on the other, or if he is lying on one side. When a person lies on one side the uppermost lung contains more air and consequently gives a more resonant note than the dependent lung.

PERCUSSION NOTES OF THORAX

The note obtained by percussion of the healthy thorax varies a great deal in different persons and in different parts of the thorax in the same person. There is no absolute standard; what is normal must be learned by experience.

The percussion sound over healthy air-containing lung is known as *normal* or *vesicular* resonance. It is a fairly long and low-pitched sound with a certain vibratory quality. Greater inflation of the lungs by deep inspiration gives *hyperresonance*, a louder, lower-pitched and more vibrant note. Over non-air-containing lung or a solid structure, percussion produces *dulness* or *flatness*, a short higher-pitched sound with feeble carrying power. Flatness designates the extremes of the qualities that characterize dulness; slight dulness is called *impaired resonance*. Over a hollow body such as the stomach when moderately distended with air one hears *tympany* or *tympanitic resonance*, a note which is high-pitched, has a characteristic timbre easy to recognize but difficult to describe and which can be produced by light percussion.

The amount of resonance obtained at any point depends on the strength of the stroke delivered as well as on the factors already indicated as affecting percussion. A powerful stroke over an infiltrated lung may bring out more resonance than a lighter blow over a normal lung. How to strike with perfect fairness and equal force upon areas being compared can be learned only by

practice. Furthermore, the distance from the observer's ear to each of the two points must be the same, otherwise the note heard from the greater distance will sound softer or duller than the same note from the nearer point.

The resonance of the various parts of the thorax can be greatly altered by a change of position, variation in the degree of muscular tension, inflation or deflation of the lungs, and certain less important factors. If, for example, the patient lies on his left side, the heart swings toward the left axilla and its dulness is extended in that direction. Resonance over the left or dependent lung will be less than that over the right or uppermost lung During deep inspiration the note will be more resonant, the margins of the lungs will be extended, and the zones of cardiac and liver dulness diminished During forced expiration the reverse is true.

In children because of the more transverse position of the heart, its area of dulness is wider; the liver, being relatively larger than in adults, has a larger area of dulness In older people the tendency for the lungs to be emphysematous gives a shade of tympany to the vesicular resonance and may make the zones of cardiac and liver dulness slightly smaller.

It is frequently helpful to compare corresponding areas on the two sides, but even so, the examiner must have in mind a definite standard for any given region. Such comparisons cannot be absolutely relied upon since normally there are slight differences in the notes on the two sides, especially where they are affected by the presence of the liver on the right and the heart on the left. In bilateral disease, the note may be altered in homologous areas so that neither can be used as a norm

Areas to be compared must be absolutely symmetrical. For example, if the sound obtained over the third rib on the right is compared with that over the third interspace on the left, a false conclusion will be drawn, because resonance is greater over an interspace than over a rib.

It is often important to determine not only the position of the resting lung but its ability to expand This can be ascertained by percussion in the following way: With the lung deflated, its lower border of resonance is carefully outlined, then the area just below this level is percussed and, at the same time, the patient is directed to inspire deeply As the lung expands and its border moves down, the note will change from dull to resonant.

The extent of the excursion varies considerably in different normal persons Usually, a movement of 4–6 cm. can be demonstrated Absence of excursion probably indicates pleuritic adhesions, pleural fluid, emphysema or some other disorder affecting free diaphragmatic excursion.

Vesicular Resonance

Anteriorly vesicular resonance is found extending downward from the clavicles toward the costal margins except where it is replaced by a dull or flat note due to presence of heart or liver. Above the clavicles are found zones of somewhat lesser resonance corresponding to the pulmonary apices In Fig 10 6 normal

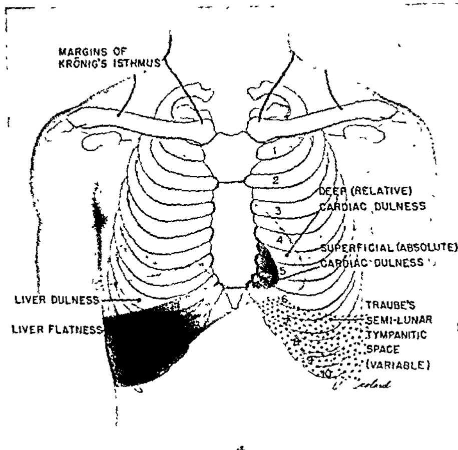


FIG 10 6 Percussion outlines of normal chest

resonance is indicated by lack of shading, diminished resonance or dulness by light shading, and flatness by heavy shading.

On the right anteriorly as one percusses downward over a normally ventilated lung, a zone of dulness is found extending from the fourth to the sixth intercostal space Here the lower border of the lung overlaps the liver, so resonance is impaired. From the sixth space to the costal margin the liver is in immediate contact with the chest wall, hence the note is flat As the percussor approaches the axilla, the line of pulmonary resonance is found to slope downward, crossing the eighth rib in mid-axillary line Posteriorly it reaches the spine above the eleventh rib or a little higher, the guide being the spine of the tenth thoracic vertebra The lowest part of the dulness is in mid-axillary line or just posterior to it, from that point toward mid-back, the border is virtually horizontal or rises slightly.

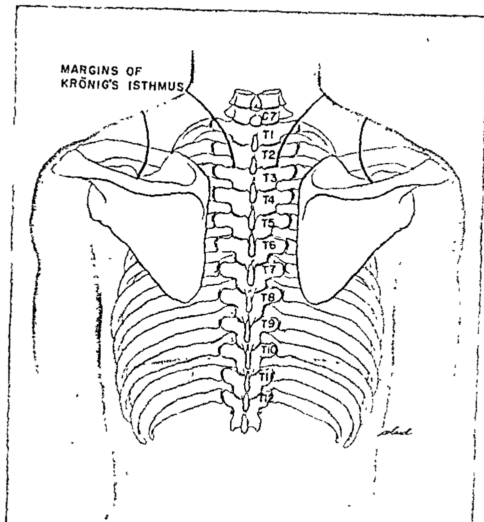


Fig 10 7 Kronig's isthmus posteriorly

On the left anteriorly, pulmonary resonance is encroached upon by the cardiac dulness described below. Anteriorly and laterally the lower border of resonance may be slightly lower than on the right but is less easily definable since it merges with tympany referred from the stomach Posteriorly it is at the same level as on the right

The superior margin of each lung extends 2–3 cm above the clavicles, producing *Kronig's isthmus*, a band of resonance usually 4–6 cm. wide across the shoulder. Its narrowest point is at the top of the shoulder, from here it spreads out anteriorly and posteriorly to merge with wider areas of resonance below the clavicle and spine of the scapula It is bounded medially by the structures of the neck and laterally by those of the shoulder Fibrotic contraction or infiltration of the pulmonary apex will narrow or obliterate this zone of resonance.

Posteriorly vesicular resonance is elicited from the apices to the lower borders It is clearest below the scapulae and between them and the spine; except in persons with heavy musculature the note in these regions is comparable to that

observed anteriorly. Above and overlying the scapulae resonance is less because of the thicker muscles. If the shoulder blades are drawn laterally by having the patient place each hand on the opposite shoulder, the interscapular zones of clear resonance will obviously widen

Normal Dull Areas

Liver Dulness and Flatness. On the right, as indicated above, the thorax shows dulness anteriorly below the fourth interspace and flatness below the sixth rib The line of flatness extends from the sixth rib at the sternum around to the level of the tenth thoracic spinous process posteriorly; the dulness shades into the flatness as one percusses into the axilla

Cardiac Dulness. *Whenever the heart borders or the supracardiac region are being mapped out by percussion, one should start in each interspace well out toward the axilla and percuss toward the sternum, observing the point at which the note changes from resonant to dull.*

The right auricle extends 1–2 cm. to the right of the sternal edge at the level of the third and fourth interspaces Theoretically percussion here should reveal a narrow band of dulness. Actually, it is obscured by resonance transmitted from adjacent pulmonary tissue and accentuated by the sounding board effect of the sternum Consequently one cannot depend on percussion measurements of the normal right cardiac border. If well-defined dulness is found just to the right of mid or upper sternum, it is most likely an indication of right auricular enlargement, pericardial effusion, displacement of the heart to the right, or some regional pleuropulmonary disorder

To the left of the sternum there is a definite area of dulness corresponding to the heart On light percussion a small zone, as indicated in Figure 10 6, gives a note which is nearly flat This is known as the area of *superficial* or *absolute* cardiac dulness and is of no clinical importance, it merely represents that part of the heart which is not covered by lung Stronger percussion will bring out a note of lesser dulness known as *deep* or *relative* cardiac dulness; this represents the size of the heart. Its left border begins in the third interspace 2–4 cm to the left of midsternum and extends downward and outward, being, in the fifth intercostal space, 7–9 cm to the left of midsternal line and on or just medial to midclavicular line This measurement is the single, most important determination as it provides the best index of cardiac enlargement or left-sided displacement

Supracardiac Dulness Resonance transmitted from the air passages and adjacent lung tissue makes percussion of the deeply seated aorta and superior vena cava notoriously inexact. Occasionally one can detect between the cardiac base and the lower border of the first costal cartilage a *zone of slight dulness* 5–6 cm wide, produced by the underlying great vessels, but this is always *uncertain* Percussion here is of value only when dulness is readily discernible and especially if the zone is wider than 5–6 cm It then indicates some abnormality such as dilatation or aneurysm of the aorta, substernal goiter, persistent thymus or mediastinal tumor.

Splenic Dulness. Accurate percussion of the spleen requires long experience.

Over the normal spleen, the trained observer may be able to map out in the lower part of the left axilla a small oval zone of dulness bounded by the ninth and eleventh ribs However, it is often obscured by tympany transmitted from air-containing stomach or colon, resonance from left lung or, less often, by dulness due to solid matter in stomach or colon or a pathologic condition in the left pleural cavity. If the spleen is moderately enlarged, dulness is often more distinct and covers a larger area but here too it may be poorly defined because of transmitted sounds.

Percussion of the spleen has its greatest value in detecting enlargement not sufficient to make the tip palpable or when perisplenic adhesions prevent the enlarged organ from descending below the costal margin on deep inspiration. X-ray examination is often more informative When the spleen is greatly enlarged, dulness is obvious but percussion is of less importance because the organ is readily palpable

Tympanitic Sounds

Tympanitic Resonance. This sound is heard by percussion over a hollow body such as the stomach when moderately distended with air It is higher pitched than normal pulmonary resonance, can be elicited by lighter percussion, and differs qualitatively from pulmonary resonance in a way easy to appreciate but difficult to describe Tympany is obtainable over most of the normal abdomen Over the thorax it can usually be found to the left of the ensiform cartilage and for a few centimeters lateral to this point over the area corresponding to that of the stomach bubble as seen by x-ray This tympanitic zone, known as *Traube's semilunar space*, varies a great deal in size according to the contents of the stomach. Normally it is bounded on the right by liver flatness, above by pulmonary resonance and the inferior border of cardiac dulness, on the left by dulness of the spleen and adjacent structures, and below, at times, by the sound over the transverse colon, which is also tympanitic, although its pitch is different owing to the different size and shape of the intestine. If the stomach is distended with gas, resultant tympany over the upper left abdomen and lower left chest will make Traube's space indefinable, if the stomach is distended with fluid or solid material the whole region will be dull or flat. Traube's space is reduced or obliterated in the presence of enlargement of the left lobe of the liver or large pericardial or left pleural effusion When the spleen is greatly enlarged Traube's space will be partially or totally obliterated, when it is only slightly or moderately enlarged it does not extend medially far enough to affect this zone.

Amphoric Resonance. Occasionally obtained over pneumothorax or a large pulmonary cavity, amphoric resonance resembles tympanitic resonance but is low-pitched and has a more hollow sound. It may be imitated by percussing the cheek when it is moderately distended with air

Cracked-pot Resonance. When percussing the chest of a crying child, one sometimes notices that the sound elicited has a peculiar *clinking* quality, like that produced by striking one coin with another but more muffled. The sound

may be more closely imitated and the mode of its production illustrated by clasping the hands palm to palm so as to enclose an air space which communicates with the outer air through a narrow chink, and then striking the back of the under hand against the knee. When the blow forces air out through the chink, a characteristic sound is heard

In disease, the cracked-pot sound is usually produced over a superficial and empty pulmonary cavity from which the air is suddenly expelled by the percussion stroke. It is better heard if, during percussion, one listens with a stethoscope, the chest piece of which is held by the patient in front of his open mouth.

Bell Tympany. This is a clear-cut, ringing, bell-like note brought out by the *coin test* Using the flat surface of one silver coin as pleximeter and the edge of another as plexor, one percusses the front of the thorax while listening with the stethoscope over the back on the same side or *vice versa*. When heard, bell tympany is an important sign of pneumothorax, but it is not present in all cases. It is not heard over normal lung or any other intrathoracic disorder.

SENSE OF RESISTANCE

By percussion one derives as much or more information from what he feels as from what he hears He gauges the degree of resistance and of vibration The resistance varies inversely with the degree of resonance. Normally a certain elasticity is felt over the lungs, it is more pronounced where resonance is greater, less evident over heavily muscled areas, and much less so over the liver and heart It is most marked in the abdomen. Vibration is also appreciable in resonant and tympanitic zones, absent in very dull and flat zones.

Over solid organs or where air has been replaced by fluid or solid material there is a dead, woodeny feeling resembling that obtained by percussing the thigh. This sense of resistance is particularly helpful in the diagnosis of pleural fluid or large, solid tumor of the lung

11

AUSCULTATION OF LUNGS

Auscultation, like percussion, can be performed by the *direct* or by the *indirect* method. In the former, one places his ear directly against the body surface; in the latter the stethoscope is used

Stethoscopic auscultation which brings out greater detail at localized points is the procedure of choice The direct method is less useful because of difficulty in reaching the supraclavicular and upper axillary regions, of comparing symmetrical areas, and of localizing the sounds heard. However, the unaided ear may occasionally perceive sounds, particularly those which are faint, high pitched and blowing, that escape detection with the stethoscope Distant tubular breathing or a soft blowing diastolic murmur may be heard only in this manner. Consequently direct auscultation should be tried when other clinical findings suggest that some such sound has been missed on indirect examination.

USE OF THE STETHOSCOPE

The binaural stethoscope with which everyone is familiar is the instrument preferred in this country. Other types are available but rarely used For the sake of comfort and to reduce distraction by extraneous noises, one should select a stethoscope which properly fits the auditory canals The usual mistake is to have too strong a spring and ear tips which are too small

The two most popular chest pieces are (1) the bell type—an elongated, bell-shaped, open receiver of hard rubber or metal about 2 5 cm in diameter, (2) the Bowles type—a shallow, metal cup about 4 5 cm in diameter, over the mouth of which is a hard rubber or celluloid diaphragm Either type will detect the usual heart or breath sounds, but because each has certain advantages, the physician should be equipped with both The bell-shaped receiver is particularly useful for examining the apices of the lungs and other localized areas and, in thin subjects, the spaces between ribs It will often pick up the faint, rumbling diastolic murmur of mitral stenosis not audible with the Bowles type With the latter one can often hear faint, higher-pitched sounds not readily heard with the bell-shaped type· the soft-blowing murmur of aortic insufficiency, for example, or the faint, high-pitched respiratory note of a deep-seated pneumonic process. The Sprague chest piece combines the bell and Bowles receivers in a single unit

Physicians with impaired hearing can obtain a stethoscope equipped for

electronic amplification; it has no special merit for those with normal hearing. For teaching purposes, some clinics use a device with multiple ear sets and electronic amplification

The stethoscope should be carefully examined from time to time to make sure that the tubes are not plugged and that there are no loose joints, or cracks in the diaphragm, either of which will produce cracking, snapping noises easily confused with rales. Care should be taken to apply the ear pieces properly. Their incorrect application is a frequent source of trouble to the beginner, who makes the mistake of introducing them into the ear so that they point downward instead of upward For the patient's comfort and to prevent introduction of muscle sounds produced by shivering, the receiver should be warmed. In comparing homologous areas, the examiner must keep his head at the same point so that the influence of extraneous sounds on those heard through the stethoscope will always be the same

The art of using a stethoscope successfully depends upon two factors: first, a knowledge of what to disregard and second, a selective attention or concentration upon those sounds which we know to be of importance.

Adventitious Sounds. A quiet room is the first essential Some experienced observers have, by long practice, schooled themselves to be oblivious of extraneous sounds in the room or outside, but even so there is less chance for error if there is perfect stillness. Other sources of distraction should be eliminated The auscultator's position should be comfortable, so that his concentration will not be disturbed. Many physicians shut their eyes while listening, so as to avoid the distraction of visual impressions

In subjects whose ribs stand out above the level of the interspaces, thus forming gaps through which external sounds may enter, a bell-shaped chest piece, sufficiently small to be inserted between the ribs, should be used. More immediate sounds, such as those produced by friction of the examiner's finger against the stethoscope or of the stethoscope against the subject's skin or hair, should be excluded The first two simulate pleural friction and can be avoided by grasping the chest piece tightly and pressing it firmly against the patient's body Sounds made by hair simulate rales; these can be eliminated by wetting the hair

Another group of sounds which the listener must learn to disregard are those produced by contraction of regional muscles, due to tenseness, chilliness, or nervousness They are also heard at the end of forced inspiration, owing to the contraction of voluntary muscles, and appear over the pectorals anteriorly or the trapezius posteriorly Muscle sounds are frequently mistaken for rales The latter, however, are more clean-cut, have a more distinct beginning and end, seem nearer the ear, and have a more crackling or bubbling quality Muscle sounds are muffled, distant, and rumbling, sounding sometimes like a continuous low pitched roar or drumming, sometimes like the slow dropping of a stream of water on a sheet of metal. Variation in the intensity or quality of a muscle sound can be produced by shifting the patient's position in such a way

as to change the stress on the regional muscles True rales do not change with shift of position

Many beginners do not listen long enough in any one place. Concentrating at one point on heart beat after heart beat or breath sound after breath sound can be very helpful If the sounds are indistinct, it is especially important to keep on listening *and to fix the attention successively on each of the different* elements in the sounds under consideration.

Position of Patient. Auscultation of the lungs is performed most satisfactorily if the patient is physically and mentally relaxed. He should be sitting or standing; when the back is being examined he should assume the slumped posture described under *Percussion* If he is too sick to sit or stand and must be examined while recumbent, he can be turned from side to side or the Bowles chest piece can be slipped under the back In either event, only the more obvious changes in breath sounds can be detected When he is on his side, comparison of homologous areas is unsatisfactory When the chest piece is under the patient, adventitious sounds such as the scratching of bedclothes confuse the listener

Manner of Breathing. The sounds produced by normal quiet breathing are not clear enough to be dependable Reliable observations can be made only if breathing is forced, deep, and through the mouth, not through the nose *The proper manner of breathing must be demonstrated by the physician before he begins to listen* Even then, difficulties may be encountered·

1. The patient may continue to breathe through his nose

2 He may hold his mouth open and go through the motions of deep respiration but actually inspire little air In these instances the sounds will be indistinct

3 *He may blow out his breath so forcibly and noisily that sounds originating* in his mouth will reach the examiner's ears directly and so alter those coming from the lungs through the stethoscope as to make them appear tubular or asthmatic when actually they are normal Differentiation of these sounds from those produced by actual disease within the thorax can be achieved by raising the stethoscope from the thoracic wall and listening while the patient continues to breathe, if the same sounds are still heard, it is obvious that they are originating in the mouth and not in the thorax

4. He may be unable to take a deep breath because of pain

Sometimes a person cannot be taught to breathe in a satisfactory manner Here a useful expedient is to have him cough several times in succession or count as long as he can on a single breath; the deep inspiration which follows the cough or the last number counted is of the type desired This procedure is tedious and trying for both parties and only the inspiratory phase can be observed, but at least it is better than nothing

When the breathing, for any reason, is not of the type suitable for auscultation, caution must be used in interpreting the sounds heard.

BREATH SOUNDS IN HEALTH

Three types of breath sounds are heard in the normal chest (1) vesicular, (2) tracheal, and (3) bronchovesicular

In listening to the breath sounds *one must compare the intensity, duration, and pitch of inspiration with the corresponding features of expiration.* This comparison is of the utmost importance in judging the type of breath sounds heard *It is equally important to have constantly in mind the standards of normal respiratory sounds learned through experience*; without a clear conception of the normal, one cannot judge what is abnormal.

Distinction must be made between degrees of *loudness* and *types* of breath sounds, the former is a matter of *intensity*, the latter of *quality* The expiratory phase is the one in which variations usually are more evident; it therefore merits the most attention. Obviously breath sounds will be better heard where the chest wall is thin and musculature poorly developed

The respiratory sounds may be diagrammatically represented as in Figures 11.1 ff, the upstrokes representing inspiration, and the downstrokes expiration.

1. The length of each line represents duration of the sound.
2. The thickness of each line represents intensity of the sound.
3. The sharpness of the angle which the upstroke makes with the perpendicular represents pitch of inspiration as compared with the angle made by the downstroke with the perpendicular, which represents pitch of expiration.

Vesicular Breath Sounds

Vesicular breath sounds—those heard over the air vesicles or parenchyma of the lung—are sometimes spoken of as "breezy" because of their resemblance to the quality of sound conveyed by the swish of the wind in a clump of trees some distance away. Compared with expiration, the *inspiratory sound* is *more intense, longer*, and *higher pitched.* This type of breathing is heard over all of the lung area, except for certain small portions to be indicated later, in which there is bronchovesicular breathing *Vesicular breathing is not heard everywhere with equal intensity* It is most distinct anteriorly over the upper chest, in the axillae, and posteriorly from the scapular tips downward. Over the thin lower edges of the lung, both posteriorly and at the sides, it is fainter, though still retaining the characteristic relationship between inspiration and expiration in intensity, duration, and pitch. Diminished vesicular breathing is simply vesicular breathing of a quieter type (*see* Fig 11.1).

Breath sounds are of normal quality but louder (*exaggerated* or *intensified*) when respiration is increased, as by exercise. This variation is also observed in unaffected areas when, because of disease, large segments of pulmonary tissue

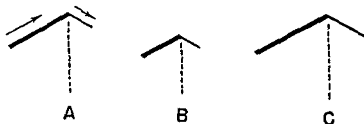


Fig 11 1 Vesicular breath sounds A Normal B Diminished. C Exaggerated.

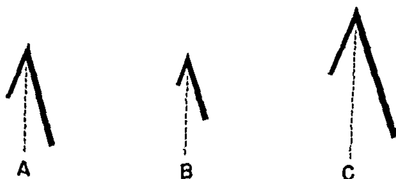


FIG 11 2 Bronchial breath sounds A Moderate intensity (This also illustrates tracheal breath sounds) B Diminished C. Exaggerated

are not functioning In normal children the sounds are *somewhat louder* than in adults.

Tracheal Breath Sounds

Tracheal breathing (Fig 11.2) is characterized by *increased duration of the expiratory sound* (increased length of the downstroke), *greater intensity of both expiratory and inspiratory sounds* (greater thickness of both lines), and *higher pitch of both sounds* (the sharper pitch of the gable on both sides of the perpendicular). The sounds have a definite tubular quality The expiratory note slightly exceeds the inspiratory in intensity, is slightly higher in pitch, and is of considerably longer duration—the reverse of the relationship in vesicular breathing. There is usually a slight interval between the sounds of inspiration and expiration.

Tracheal sounds are normally heard over the trachea and larynx and sounds closely resembling them are heard over the lower cervical vertebrae

Bronchial breath sounds, also called tubular sounds, which are heard in disease, are very similar to tracheal breath sounds, but are more harsh and possibly somewhat lower in pitch The resemblance is sufficiently close for tracheal sounds to be used as standards by which to judge bronchial breath sounds.

Bronchovesicular Breath Sounds

Bronchovesicular breathing is a mixture of bronchial and vesicular elements. It is at once obvious that a wide range of sounds is encompassed by this term and that the sounds heard depend on which element predominates As a general rule, expiration is *more intense, longer,* and *higher pitched* than inspiration In some cases, the two phases will be quite similar in intensity, duration, and pitch, but as the bronchial type is approached, the expiratory phase heard in true bronchial breathing will tend to predominate Some authorities distinguish between bronchovesicular and vesiculobronchial breathing on the basis of the more prominent element, but this distinction is of no particular importance

Two of the common types of bronchovesicular breathing are illustrated in

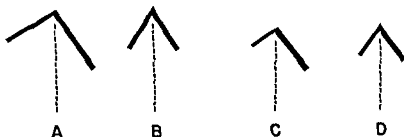


Fig 11 3 Bronchovesicular breath sounds A and B Two common types C and D Same, diminished

Fig 11 4 A Anterior location of normally heard tracheal and bronchovesicular breath sounds

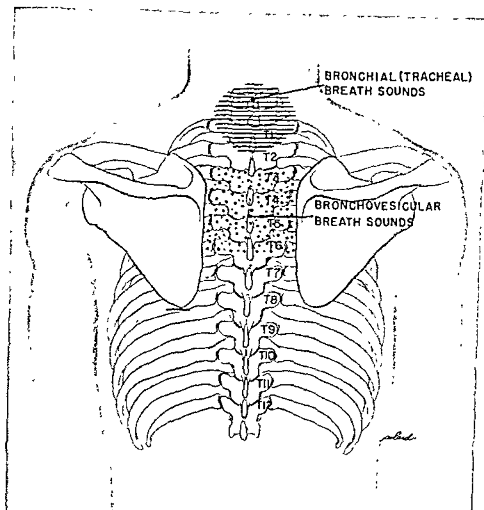


Fig 11 4 B Posterior location of normally heard tracheal and bronchovesicular breath sounds

Figure 11 3 Bronchovesicular breath sounds, like vesicular and bronchial breath sounds, can be either more or less intense than normal

In the normal chest bronchovesicular breathing is audible where normal lung tissue overlies the large bronchi, viz., anteriorly over the upper end of the sternum and just beside it at the level of Louis's angle, and posteriorly over the interscapular spaces at the level of the third and fourth thoracic vertebrae. In these areas the bronchial element predominates Bronchovesicular breathing, with the bronchial element much less predominant, is observed in some normal persons at either or both apices, more commonly the right. In such instances it is limited to the area extending from the clavicle or a few centimeters below it over the shoulder, to the spine of the scapula When present it does not necessarily indicate disease. To determine its significance, one must rely on other clinical findings and roentgenologic examination. Bronchovesicular breathing, if heard in areas of *the thorax where vesicular breathing is normally present, is* an indication of disease.

BREATH SOUNDS IN DISEASE

Disease of the respiratory system may cause alteration of the normal breath sounds or the appearance of sounds never heard in the normal chest.

VESICULAR BREATH SOUNDS

Exaggerated Vesicular Breathing

Normally in children, in adults with thin, flexible chests, and under circumstances such as exertion which leads to deep breathing, the intensity of breath sounds is increased over both lungs. Exaggerated vesicular breathing may occur in one lung or part of a lung as a compensatory process (*compensatory* or *complementary* breath sounds) It is heard, for example, over the whole of one lung when function of the other is hampered by pleuritic adhesions, marked deformity of the chest wall, as in severe spinal curvature, pressure of an accumulation of air or fluid in the pleural cavity, obstruction of a bronchus, or solidification in such disorders as tuberculosis, pneumonia and malignant disease. If the function of parts of one or both lungs is diminished by disease, the uninvolved areas of one or both may show the same variant.

Diminished Vesicular Breathing

The causes of decreased intensity of breath sounds without change of quality are numerous The more common ones are mentioned here in an order corresponding roughly to their relative frequency

Fluid, Air or Solid Tissue in the Pleural Cavity. Probably the commonest cause for diminution or absence of normal breath sounds is an accumulation of fluid in the pleural cavity resulting from inflammation of the pleura (*pleurisy with effusion*), or from transudation (*hydrothorax*) The fluid interferes with pulmonary expansion in the affected area so that little or no vesicular murmur is produced, and hence none is transmitted to the ear Air in the pleural cavity (*pneumothorax*) will have the same effect Thickening of the parietal or visceral pleura or a malignant growth of the chest wall may, by hampering pulmonary movement or interfering with transmission of sound, render the breath sounds feeble or inaudible without, as a rule, any modification of type One important exception to the above must be mentioned breath sounds—usually bronchial—are sometimes heard over a large pleural effusion

Pathologic Conditions within the Lung. In the earliest stages of pneumonia the breathing in the affected area may be feeble or nearly suppressed Pulmonary edema may diminish the breath sounds In emphysema the sounds are faint and usually show variation in quality.

Restriction of Respiratory Movement. The most likely causes are

1 Thoracic pain such as occurs with acute pleuritis, arthritis of the spine, or fractured rib

2 Interference with normal movement of the diaphragm by upward pressure from some abdominal disorder such as ascites or gaseous distention.

3 Paralysis of the respiratory muscles Alternation of sounds will be uni- or bilateral, depending on whether respiratory movement is impaired on one or both sides.

Interference with Flow of Air into Lungs. The more common causes are.

1 Spasm or edema of the glottis, obstruction of the trachea, or other disorder in the upper respiratory tract restricting free flow of air. Breath sounds will be decreased on both sides.

2. Total or partial occlusion of a bronchus. The whole or part of a lung may be involved, depending on the location of the lesion.

3 Bronchitis or asthma. Ventilation and consequently breath sounds may be diminished; the change may be overlooked because one's attention is so readily distracted by the associated rales (*see below*).

Bronchial Breath Sounds

The distinction has already been made between the tracheal breath sounds heard in the normal subject and bronchial breath sounds of disease Bronchial breathing is not heard over normal lung but is frequent in pulmonary disease, usually as an indication of solidification or marked infiltration Depending on the nature of the ailment and the location and size of the area involved, it will be intense, moderate or diminished. It occurs in the disturbances listed below, provided the bronchi leading to the affected area remain open

Pneumonia. The most common cause of bronchial breathing is lobar pneumonia. In bronchopneumonia, it may be present unless the patches of pneumonic solidification are too small to transmit bronchial sounds to the surface. As a rule, it is heard in atypical pneumonia only when pulmonary involvement is extensive.

Tuberculosis. Bronchial breathing may be present if there is an area of marked pulmonary infiltration or of tuberculous pneumonia. It also occurs as a result of compression atelectasis in some cases of tuberculous pleurisy with effusion.

Pleural Fluid. Bronchial breathing is heard in about a third of these cases Usually it is diminished or distant, occasionally moderate or loud. *Loud bronchial breathing is especially common over fluid or pus appearing in a pleural cavity during or after an attack of lobar pneumonia* This is an important point to remember, for the observer unfamiliar with this fact may fail to diagnose postpneumonic empyema or effusion by erroneously assuming that solidification alone is the cause of the bronchial breathing

Compression Atelectasis. Any disorder causing compression of a portion of a lung such as pleural effusion at the base, high diaphragm from upward pressure in the abdomen, or large pericardial effusion will cause bronchial breathing over the compressed area, unless the region affected is so small that vesicular breath sounds from surrounding unaffected lung tissue obscure the bronchial sounds.

Obstructive Atelectasis. In rapidly developing obstructive atelectasis due to a cause such as foreign body inhalation or retention of bronchial secretion, the inflammatory reaction in the affected portion of the lung may be sufficient to cause bronchial breathing, provided some air is passing the obstruction

Pulmonary Infarction. Engorgement of the affected area produces solidifica-

tion not unlike that of early lobar pneumonia so that bronchial breathing may be heard unless the infarction is small. The abnormal sounds may not appear for several days after the embolic episode.

Less commonly, bronchial breathing is found in other pulmonary disturbances such as abscess, chronic fibrosing pneumonitis, malignant disease, and severe congestion.

Bronchovesicular Breath Sounds

Any of the disorders mentioned under bronchial breath sounds, but usually in a less advanced stage, may give rise to areas of bronchovesicular breathing In such cases, a smaller area of lung is involved and the bronchial breathing, transmitted from the affected area, is modified by the vesicular sounds of surrounding normal tissue Or there may be incomplete solidification over a larger area, causing a mixture of the two types of sound

Asthmatic Breath Sounds

In asthmatic breathing inspiration is either normal or a short gasp, while expiration is much prolonged, usually high pitched, and wheezing in character. Usually there are associated musical rales. Asthmatic breathing may be more intense or more feeble than normal It occurs with bronchial asthma and in some cases of empysema and bronchitis In severe emphysema the breath sounds may be barely audible.

Other Abnormal Breath Sounds

Cavernous Sounds Cavernous breathing is characterized by a deep hollow quality and low pitch, with expiration lower pitched than inspiration, just the opposite of bronchial breathing It may be heard over a cavity or an area of pneumothorax, but its presence is not essential for the diagnosis of either.

Amphoric Sounds. These resemble cavernous sounds but are more high-pitched and have a certain metallic quality When heard, they indicate a cavity or area of pneumothorax but are not necessarily present with either

Cog-wheel Sounds. The inspiratory and occasionally the expiratory phases are interrupted by a series of short, irregular pauses. This variant is usually

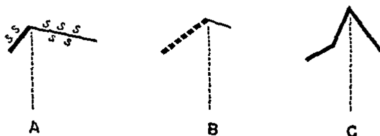


Fig 11 5 Abnormal breath sounds A Asthmatic (Letters "S" represent sibilant or sonorous rales) B Cog wheel C. Metamorphosing

confined to a relatively small portion of lung, chiefly upper lobe, and is probably due to *pleural adhesions or disease of the finer bronchi slightly delaying* entrance of air into the alveoli. It is suggestive of early tuberculosis Rarely, it is heard over the entire thorax as a result of chilliness, nervousness or fatigue Cog-wheel sounds must be differentiated from *cardiopulmonary murmurs,* which have the quality of breath sounds but are synchronous with cardiac systole

Metamorphosing Sounds. In an occasional case a change will be noted in the quality or intensity of the breath sound during a respiratory cycle. One which starts feebly may become more intense, or one which is vesicular at onset may become bronchial or amphoric. A plausible explanation for this phenomenon is further opening of a partially occluded bronchus during the cycle.

Metallic Tinkle. A clear-cut, tingling metallic sound, resembling the faint tinkle of a bell, is occasionally heard during respiration over a pneumohydrothorax, *a pneumopyothorax, and, rarely, over a large partially filled pulmonary* cavity. It is thought to be due to air bubbling through the fluid from a fistula in the lung

Succussion Sounds. These are not actually *respiratory* sounds They are splashing noises which may be heard over a body cavity containing air and fluid when the patient is briskly shaken. The sounds themselves are often miscalled succussion; the term refers to the act of shaking. They are brought out by holding the stethoscope against the body wall over the affected area and giving the patient a series of abrupt shakes. Occasionally they are audible at a distance, sometimes the patient himself is aware of them. Succussion sounds are pathognomonic of a mixture of air and *fluid in the same cavity, when heard* in the thorax they usually denote pneumohydrothorax, rarely pneumopyothorax

Similar sounds commonly occur when there is a mixture of fluid and air in the stomach or a distended bowel, and are often wrongly attributed to pneumohydrothorax, especially when stomach or bowel is herniated through the diaphragm

Succussion sounds do not occur in simple pleuritic effusion, the presence of both fluid and air is required for their production.

RALES

The term *rales* is applied to sounds produced by passage of air through bronchi which contain secretion or exudate, which are narrowed by spasm or swelling of their walls, or both. Rales vary in quality, intensity, pitch and other characteristics, depending on the mechanism of their production. They are often influenced by deep breathing or coughing If not heard during respiration of normal depth they may be elicited by deep inspiration or by a voluntary cough at the end of expiration In the latter event, they are heard during the subsequent inhalation On the other hand, cough, presumably by freeing a bronchus of exudate, may cause rales to disappear As a rule, those which per-

sist during an examination or are brought out by cough are of more significance than those which disappear following a few coughs

Moist Rales

Coarse. Loud gurgling sounds (*ronchi*) may be heard even at some distance from the patient. They occur in states of unconsciousness, great weakness or severe pulmonary edema because of inability of the patient to cough out secretions from the trachea and larger bronchi. Although connoting a serious situation they do not always portend death, as the laity believe.

Medium. Known also as *crepitant* rales, these are small, clear-cut bubbling or sometimes muffled clicking sounds due to presence of fluid or exudate in the smaller bronchi, bronchioles and perhaps the alveoli. They are most numerous during inspiration, especially toward the end of the phase They occur in bronchitis, pneumonia, tuberculosis, infarction, pulmonary abscess, gangrene, pulmonary congestion or edema, bronchiectasis, and certain less frequent disturbances.

Fine. Fine moist rales lack the bubbling quality of medium rales and are best described as and sometimes termed *crackling* They are especially apt to occur at the end of inspiration, a large number being involved in a short space of time, so that one often speaks of an *explosion* of fine crackling rales (*see* Fig 11 6) They are comparable to what one hears if he holds a group of hairs between his thumb and finger and rubs them together. To the inexperienced ear they may seem to blend into a continuous sound, but with practice the component parts can be distinguished

Fine crepitant rales are the ones most likely to be confused with the sounds heard when the stethoscope is applied to a hairy surface As noted earlier the adventitious sounds can be eliminated by thoroughly wetting the hair. When a person who has been breathing normally takes his first deep breaths at the start of auscultation, fine moist rales are likely to be heard along the pulmonary margins in the axillae and, less often, at the bases They usually disappear after the first few deep breaths, but sometimes persist Occurring more frequently in older persons and those who have been confined to bed, they are thought to be due to separation, by the active breathing, of agglutinated bronchial surfaces in parts of the lung which have become mildly atelectatic from disuse.

In disease, fine moist rales occur in the same disorders as medium moist



Fig 11 6 Explosion of fine rales at end of inspiration.

rales. A series of explosions of either the fine or medium variety *heard at the end of inspiration over a pulmonary apex is said to be especially suggestive of early* tuberculosis.

Consonating Rales. This term refers to unusually sharp and vibrant moist rales which are likely to be heard over a sizable area of solidification or a large cavity.

DRY RALES

Passage of air through bronchial tubes narrowed by mucosal inflammation, as in bronchitis, or by spasmodic contraction, as in asthma, *and usually containing* a thin tenacious exudate, gives rise to a multitude of squeaking and groaning sounds (*dry* or *musical* rales). Those in the larger tubes are deep-toned and groaning (*sonorous*), those in the finer tubes piping, squeaking and whistling (*sibilant*). As a rule they are heard more distinctly during expiration, especially *when due to asthma Musical rales are, of all adventitious sounds, the easiest* to recognize, but also the most fugitive and changeable. They appear now here, now there, shifting from time to time, perhaps totally disappearing, only to reappear shortly.

PLEURAL FRICTION

The parietal and visceral surfaces of the healthy pleura pass noiselessly over each other during respiratory movement. When they are roughened by exudate as in fibrinous pleuritis or forced against each other by unusual pressure, the rubbing of one against the other produces a characteristic sound known as pleural friction. The most common site is the lower part of the axilla, but friction may be heard anywhere in the thorax. It is sometimes widespread and *may appear over a large part of one or both lungs. The sound may* be closely imitated by holding the thumb and forefinger close to the ear and rubbing them against each other with strong pressure, or by pressing the palm of one hand over the ear and rubbing its dorsal surface with the fingers of the other.

Friction sounds are most often heard during both phases of respiration, occasionally *only during inspiration, particularly toward its end. They are usually* catchy, jerky, interrupted, irregular and appear to be close to the listener's ear. They vary grealy in intensity and quality, and may be grating, rubbing, rasping or creaking Coarse friction is spoken of as "leathery." Friction sounds sometimes temporarily disappear after the patient has taken a few full breaths, probably *because the rubbing together of the rough pleural surfaces* smooths them down After a period of superficial breathing, they will reappear Pressure exerted on the overlying thoracic wall by the hand or stethoscope may increase friction sounds To avoid rubbing sounds simulating friction, care must be taken to apply the pressure without permitting the hand or stethoscope to shift position.

In well-marked cases friction can be felt if the hand is laid over the affected area; occasionally the vibrations are so coarse that they can be heard and felt by the patient himself.

Differentiation between fine friction sounds and fine moist rales is sometimes difficult The following facts are of assistance in making the distinction:

1 A small area of pleural friction is apt to be localized in the axilla
2. Friction sounds seem close to the ear.
3 Friction is more apt to be heard in both phases of respiration.
4. Friction is increased with pressure over the affected area.
5 Friction is less apt to be altered by coughing

It is also difficult at times to distinguish pleural friction from muscle sounds. This is particularly true of the scapular region if the patient is being examined with his arms folded tensely across his chest These sounds are less jerky in character than pleural friction, increase in intensity as the shoulder joint is approached and can be abolished by having the patient shift or relax his arms.

VOICE SOUNDS

The Whispered Voice

Auscultation of the sounds created in the thorax by the whispered voice is most useful in determining the presence of pulmonary infiltration, especially when breath sounds are not satisfactorily heard or when the patient should be spared the pain or fatigue of forced breathing Whispered voice sounds are virtually equivalent to those of forced expiration but require less exertion.

The subject is instructed to repeat in a whisper such a phrase as *one, two, three* or *ninety-nine,* while the examiner listens to the various parts of the thorax and notes the character and intensity of the sounds transmitted The test is of no value unless the phrase is actually whispered--not muttered or softly spoken.

In the majority of normal persons, the whispered voice is heard only faintly, except anteriorly and posteriorly in the regions overlying the trachea and primary bronchi and in the neck posteriorly as far down as the seventh cervical vertebra Even here they are indistinct and the syllables are not clearly made out. Sometimes, especially at the bases, they are not heard at all

Whispered Pectoriloquy (Bronchial Whisper). Over an area of solidification whispered voice sounds are heard distinctly, are more intense, higher-pitched, and have a prolonged expiratory phase The larger the area affected, the more pronounced will be the changes *These variations in the whispered voice constitute a more delicate test for solidification than bronchial breathing* because they may be observed over an area too small or deep to effect appreciable changes in breath sounds They are of particular value in a patient suspected of having early pneumonia or a small zone of pulmonary collapse or infarction

Over the part of a lung compressed by and lying above the level of pleural effusion one is likely to hear bronchial whispered voice sounds; when the lung behind the fluid is completely solidified the whispered words may be clearly distinguished over the zone of fluid itself. In lesser degrees of solidification the syllables are more or less blurred.

If the whispered voice sounds, normally audible over the lower cervical

vertebrae, are heard over the upper thoracic vertebrae as well (or if the spoken *voice sounds in this region have a whispered timber*), enlargement of the tracheobronchial lymphnodes or tumor in the upper part of the posterior mediastinum should be suspected (*D'Espine's sign*) However, this variant is occasionally *found in normal children*

The Spoken Voice

Spoken voice sounds (*vocal resonance*) are tested by listening over the thorax while the subject repeatedly enunciates in natural tones some such phrase as *one, two, three* or *ninety-nine* In general, these sounds are less valuable than the whispered sounds. Normally the vocal sounds heard over the thorax are less loud and less clear than those heard over the larynx, seeming somewhat diffuse and distant. No words can be distinguished. The sounds are most audible in the interscapular region, over the upper sternum, and in the supraclavicular spaces. They are less intense in women and children than in men, whose voices are lower in pitch and therefore set up greater vibrations.

Vocal sounds are affected by the same factors that influence tactile fremitus, they are increased by solidification of the lung, decreased by intrapleural air or fluid, thickened pleura and obstruction to flow of air into the lung Vocal resonance may be increased when fluid is present in the pleural cavity, if the lower part of the lung which is in contact with the fluid is solidified as a result of infiltration or compression

Bronchophony. In some cases, solidification of lung not only increases the intensity of the spoken voice (*pectoriloquy*) but also effects a *change in its* quality, so that it sounds more concentrated, nearer the listener's ear, and the words are more clearly heard. *Bronchophony* refers particularly to the trans*mission of distinct words, not merely diffuse, blurred voice sounds* It is most common in pulmonary solidification or compression

Egophony. This is a form of bronchophony characterized by a nasal or bleating quality. The sounds heard are less resonant than in normal vocal resonance or ordinary bronchophony It is most often heard at a point somewhat below the upper level of an accumulation of fluid or pus in the pleural cavity. Some observers regard it as an important sign of such an accumulation, others believe that egophony is just as likely to occur over solidification and has little value as a differential point.

INFLUENCE OF BRONCHIAL OBSTRUCTION

The various signs described above can be entirely altered by partial or total obstruction of a bronchus resulting in interference with free flow of air to or

tactile fremitus, total obstruction by retained secretion of the bronchus leading to the consolidated area will change the signs to flatness, markedly diminished or absent breath sounds, whisper, and tactile fremitus; rales previously present will disappear. If the exudate is later coughed up and air re-enters the affected region, the original signs will reappear.

AUSCULTATION OF HEART

GENERAL CONSIDERATIONS

Auscultation of the heart is best performed by the indirect method although, in rare instances, the direct will reveal the faint murmur of slight aortic regurgitation or slight pericardial friction which is not heard with the stethoscope. The examiner should be equipped with both the bell-shaped and the Bowles receivers, as certain murmurs not audible with one may be detectable with the other. Routinely, examination is carried out with the subject both upright and recumbent; occasionally, as indicated below, other positions are necessary. We listen to the heart for:

1. Rate and rhythm of beat
2. Intensity and character of sounds.
3. Presence of murmurs or other extra sounds.

Often, we must do more than interpret what is readily heard. A deliberate search must be made for some particular murmur or sound whose presence, suggested by other clinical findings, might provide an important clue to correct diagnosis Often a variant not heard with the patient upright or recumbent may be detected by listening while he lies on one side or the other, sits with his trunk bent forward, or after he has accelerated heart beat by exercise. In doubtful cases, the phonocardiograph may be of help in detecting and evaluating heart sounds and murmurs but is not essential for routine work.

Heart Sounds. This term is applied to the sounds thought to be due to contraction of heart muscle and movements of the valves Although in disease these sounds are often abnormal the presence of normal sounds does not necessarily indicate a healthy heart *A seriously diseased heart may appear perfectly normal on auscultation*

Heart Murmurs. Extra sounds not ordinarily heard in normal hearts are known as murmurs These may be due to valvular disorders or to relatively unimportant factors Murmurs are usually, but not always, longer than the heart sounds and appear between them However, one may occur with a sound, wholly or partially masking it *Although found in many types of heart disease, murmurs may be heard in a healthy heart.* Furthermore, in certain types of cardiac disease, murmurs are not necesarily present.

Valve Areas. The entire precordium, the areas over the great vessels, and, in some cases, the surrounding regions must be examined. Attention is focused

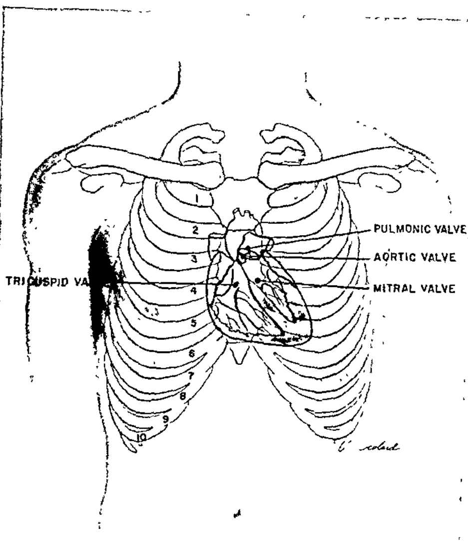


FIG 12 1 Anatomic positions of heart valves

on four sites indicated below (*valve areas*) A murmur heard in each of these regions is most likely to originate at the valve designated but the areas do not correspond to the anatomical positions of the valves.

MITRAL AREA. This is the region of the cardiac apex, normally the fifth intercostal space near midclavicular line. Most murmurs best heard here are produced at the mitral orifice

PULMONIC AREA. This is in the second left intercostal space near the sternum. Here sounds originating in the region of the pulmonary valve are most audible

AORTIC AREA. This in the second right intercostal space near the sternum Murmurs which are loudest here are produced in the region of the aortic valve. One important exception is the diastolic murmur of aortic insufficiency which is usually best heard to the left of the sternum at the level of the third and fourth costal cartilages.

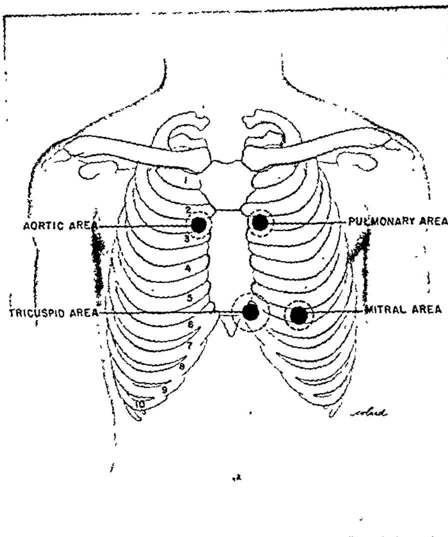


FIG 12 2 Valve areas Note that these do not conform to anatomic positions shown in Fig 12 1

TRICUSPID AREA This is the region of the ensiform cartilage. Murmurs loudest here may originate at the tricuspid orifice or elsewhere, their source is often open to question

NORMAL HEART SOUNDS

Strictly speaking, there are three sounds associated with each cardiac cycle, but ordinarily one thinks in terms of only the first and second. Except in childhood and youth, the third is usually too faint to be heard

First Sound Synchronous with cardiac systole and occurring with the cardiac impulse, this is low, dull, longer than the second sound, and has a certain booming quality There are two presumable factors in its production: (1) the

sudden closing of the mitral and tricuspid valves; (2) the systolic contraction of heart muscle.

Second Sound. Following the first after a short pause, this is higher pitched, has a more snapping quality, and is of shorter duration It is probably caused by the sudden closing of the aortic and pulmonic valves at the end of systole. Before the next first sound there is a longer pause corresponding to diastole.

Third Sound. This occurs in diastole shortly after the second sound, and is short and faint. Usually heard only in healthy young adults and children, it is of low pitch and intensity and most likely audible at the apex with the subject recumbent. It is probably due to vibration of ventricular walls and auriculo-ventricular valve cusps caused by inflow of blood to the ventricles during diastole

The *time intervals* between the various sounds vary with the heart rate: the slower the rate, the longer the intervals As the rate increases, the intervals obviously become shorter, but the decrease in the diastolic interval—that between the second and the first sound—is relatively greater than the change between the first and second sounds

The *quality* of the sounds and their *time relationship* make it possible for the experienced observer to recognize a definite rhythm and thus easily distinguish between the first and second sounds This distinction is of the utmost importance, first, because it facilitates appreciation of changes in the sounds themselves, and second, because it serves as a guide to the timing and identification of murmurs Those who are not sufficiently experienced to distinguish the sounds by their rhythm and quality can best identify them by feeling or looking at the cardiac impulse while listening, the first sound is synchronous with the impulse. If the apex impulse cannot be seen or felt, the pulsation of the carotid artery can be used as a guide, although it is unsatisfactory when the heart rate is rapid because of the slight difference in time between the first sound and the carotid pulsation Use of a more distant artery for this purpose leads to error because of the time it takes the pulse wave to reach the periphery. Rapid heart action sometimes obscures the quality and time relationship of the two sounds, so that even the experienced observer may have to be guided by the impulse

The intensity and quality of the heart sounds vary in the different areas At the apex the first sound is definitely louder and more booming than the second; it gives the impression of being accented like the first syllable of a trochaic rhythm At the base of the heart the accent changes, the stress falling on the second sound, which is more distinct than at the apex, the first sound seems less intense but remains duller and longer than the second The second sound may be louder over the pulmonic area than over the aortic, or *vice versa*, the difference depending on conditions which will be discussed later Over the tricuspid area the sounds are essentially the same as those heard at the apex, but less intense.

In patients with thick chest walls or pronounced emphysema, the heart sounds may be poorly heard, sometimes almost inaudible. They may come through more clearly if the patient bends far forward or lies on his side and is examined with the lungs at the point of maximum expiration In young persons with thin, elastic chests, the heart sounds are heard with greater intensity than in older subjects whose chest walls are thicker and stiffer. If one or both lungs are retracted by disease, the sounds heard over an area of the heart not covered by lung (as it normally is) will seem intensified This is more apt to occur at the base, and, when it does, pulsation of the pulmonary conus in the second left interspace or of the ascending aorta in the second right interspace may be seen or felt. *Before attributing abnormal heart sounds to disease of the heart such factors as these must always be excluded.*

MODIFICATIONS OF HEART SOUNDS

Exertion, excitement, thyrotoxicosis, and other states increasing cardiac activity are apt to cause accentuation of both heart sounds. Physical or mental repose or any disorder such as obesity, emphysema or pericardial effusion which interferes with transmission of the sounds, will reduce their intensity. Neither accentuation nor diminution necessarily indicates heart disease. In certain pathologic states both heart sounds are modified, in others, only one.

The First Sound

Accentuation. In addition to the causes mentioned above the first sound may be intensified by.

1 Cardiac hypertrophy prior to onset of failure.

2 Mitral stenosis. In mild mitral stenosis, the heart sound is slightly louder than normal, in moderate or severe stenosis it is very characteristic—intense, sharp and slapping or snapping.

3. Disordered cardiac rhythm All of the beats may be louder than normal in paroxysmal tachycardia or auricular flutter. Some of the beats are intensified in premature beats, auricular fibrillation and certain cases of heart block

Diminution. This is found most commonly in·

1. Myocardial weakness from any cause. In serious cases, the weak first sound may resemble the second, so that, if the rate is rapid, tic-tac rhythm is produced (*see below*) This is a bad sign.

2. Peripheral circulatory failure

3 Prolonged auriculoventricular conduction time. Here closure of the mitral and tricuspid valves is less sudden so that the first sound is diminished.

Variation from Beat to Beat. In disordered cardiac rhythms, such as premature beats, auricular fibrillation, and heart block, the first sound varies in intensity. Premature beats may be louder or softer than normal beats, sometimes so faint that they cannot be heard at all Untreated auricular fibrillation usually shows wide variation in intensity from beat to beat Differences exist but are less pronounced when the fibrillation is well controlled. In complete

heart block when an auricular contraction happens to fall just ahead of or simultaneously with a ventricular contraction, the resultant first sound will be louder than the others

Reduplication. Many healthy hearts show doubling of the first sound in the region of the apex impulse; the effect produced is suggested by pronouncing the syllables "*turrupp*" or "*trupp*." This must be carefully differentiated from gallop rhythm and presence of third heart sound. A similar varation is likely to be heard in mitral stenosis, auriculoventricular block, and bundle branch block.

The Second Sound

As a rule, the pulmonic second sound is louder than the aortic in youth; in middle age the two sounds are of approximately equal intensity; in old age the aortic is louder. Pathologic modification of the second sound may be limited to either the pulmonic or the aortic area or, less often, involve both

Decision as to whether the pulmonic second sound is normal or modified must be based on one's knowledge gained by experience of what is normal *The age of the patient and other factors noted above which affect both sounds* must be taken into consideration. The same applies to the aortic second sound Comparison of the second sound in the two areas is also helpful For example, *if, in a younger person, the aortic second sound is louder than the pulmonic,* increased pressure in the systemic circulation is to be suspected, if, in an older person or one with systemic hypertension the pulmonic is louder than the aortic, one would suspect pulmonary hypertension secondary to a *failing left* ventricle

Accentuation of Pulmonic Second Sound. This indicates increased pressure in the pulmonary circulation which, if chronic, will eventually lead to hypertrophy of the right ventricle If, because of the added strain, the right ventricle fails, the pulmonic second sound may become diminished. The most common causes are.

1. Mitral stenosis or insufficiency
2. Left ventricular failure from any cause.
3. Certain bronchopulmonary disorders, such as extensive lobar pneumonia, emphysema and chronic pulmonary fibrosis in which circulation in the pulmonary circuit is impaired
4. Pulmonary embolism, moderate or large.
5. Certain congenital cardiovascular defects

Diminution of Pulmonic Second Sound. When the right ventricle fails from any cause, the pulmonic second sound will be less intense than normal It may be faint or absent in pulmonary stenosis.

Accentuation of Aortic Second Sound. Pathologic accentuation of the aortic second sound is caused by:

1. Systemic hypertension from any cause
2. Loss of aortic elasticity, as in syphilitic aortitis or in arteriosclerosis of the aortic wall or valves

Diminution of Aortic Second Sound. The usual causes are:

1. Any disturbance which diminishes the amount or velocity of blood forced into the aorta by the left ventricle, the most common being: (a) aortic stenosis or mitral stenosis, although either of these may exist without diminution of the aortic second sound; (b) weakening of the left ventricle; (c) peripheral circulatory failure; (d) weak premature beats or some of the weaker beats in auricular fibrillation. Some of these beats may be so weak that they fail to open the aortic or pulmonic valves at all, and the second sound is then absent.

2. Systemic hypotension. This is usually due to severe circulatory failure, severe anemia or some debilitating disease.

3. Disease of the aortic cusps, preventing their proper closure. In advanced cases, valve closure may be so seriously interfered with that the aortic second sound is entirely absent.

Accentuation of Both Second Sounds. This will be present if there is increased tension in both the peripheral and pulmonary circulations.

Reduplication of Second Sound. This is more common than reduplication of the first sound. If the lungs are at the point of full expiration, reduplication of the second sound can sometimes be heard in the pulmonic area as a normal variant. Its common pathologic causes are:

1. Pulmonary hypertension from any cause.
2. Systemic hypertension. In these instances the increased pressure on one side or the other probably causes earlier closure of the corresponding valve.
3. Bundle branch block.

The Third Sound

As already indicated, the third sound is a common finding in children and young adults but is usually not heard in older subjects. It may be audible in constrictive pericarditis. The extra sound of protodiastolic gallop rhythm is similar to the third heart sound in quality and time; the two can be differentiated only by evaluating other features of the case.

Tic-Tac Rhythm

If the diastolic pause is shortened by rapid rate and the apical first sound loses its booming quality because of myocardial weakness, the first and second sounds resemble each other in quality and have a cadence similar to the ticking of a clock. This rhythm, comparable to that heard in the fetus, is sometimes called *embryocardia*. Occurring in myocardial weakness from any cause and in peripheral circulatory failure, it is regarded as a serious sign.

Gallop Rhythm

An extra sound is clearly heard during each cardiac cycle, causing a characteristic three-beat cadence resembling the hoof-beats of a galloping horse.

When the heart rate is slow, the experienced observer may be able to distinguish several different types. The extra sound is heard, (1) just after the second heart sound (*protodiastolic*); (2) in mid-diastole (*mesodiastolic*); (3) at

the end of diastole (*presystolic*); and (4) between the first and second sounds (*systolic*). Ordinarily the exact type of diastolic gallop rhythm cannot be distinguished, since the intervals between sounds are too short. It is possible only to recognize that diastolic or systolic gallop rhythm is present; this is sufficient for clinical purposes.

Diastolic Gallop Rhythm. This is best heard at the apex or to the left of lower sternum Sometimes the abnormal rhythm may be better appreciated by palpation than auscultation When, as often happens, the extra sound in diastolic gallop rhythm cannot be distinguished by auscultation from the normal third heart sound, differentiation is based on the circumstances under which it is heard The normal third sound occurs without organic heart disease It is most common in youth, especially following exertion, and in cases of neurocirculatory asthenia The abnormal third sound of gallop rhythm is found in conjunction with other signs of cardiac disease.

Diastolic gallop rhythm almost always indicates serious weakness of the heart muscle and, as a general rule, is a grave prognostic sign It is encountered most commonly in disorders causing ventricular strain. If heard or felt best near the apex, left ventricular strain is indicated, if just to the left of the sternum, right ventricular strain.

Systolic Gallop Rhythm. The extra sound occurs between the first and second sounds, is best heard at the apex, and disappears as one moves the chest piece toward the base. This variant is rare and has no clinical significance.

Auricular Heart Sounds

Auricular contraction may cause reduplication of the first sound in cases of slight auriculoventricular block. With complete auriculoventricular block the sounds may be heard at varying intervals between those of the ventricular contractions If present, they are best heard between the cardiac apex and lower end of the sternum or over either auricle.

Adventitious Heart Sounds

A peculiar scratching, leathery or snapping sound is occasionally produced by the normal heart during systole It has no significance

A metallic quality of the heart sounds is sometimes heard in cases of pneumothorax or gaseous distention of the stomach or bowel

Tinkling or splashing sounds synchronous with heart beat are in rare instances heard in pneumothorax, hydropneumopericardium, and herniation of the stomach through the diaphragm

Peculiar crunching sounds are produced over the precordium by each heart beat in pneumomediastinum (*see* Chap. 28).

SOUNDS OVER PERIPHERAL VESSELS

The normal heart sounds are heard over the subclavian and carotid arteries in adults. In the young, only the second sound is usually audible Over the jugular veins one can occasionally hear three very faint sounds coinciding with the waves seen in the jugular pulse.

A systolic sound can be heard over the femoral artery in about 7 per cent of normal persons With this exception, systolic sounds appear over the large arterial vessels in normal persons only if their walls are partially compressed.

In free aortic regurgitation a systolic sound is almost always audible over the femoral, usually over the brachial, and sometimes over the radial arteries. Over the femoral artery it may be so intense as to be called *pistol-shot* sound. Other disorders with a high pulse pressure, such as thyrotoxicosis and high fever, may show this phenomenon but it is not as loud as in free aortic regurgitation

HEART MURMURS

Murmurs are extra sounds heard over the heart They occur in a variety of circumstances described in the following pages They may be whistling, rolling, rumbling, blowing or piping, but in nowise do they resemble a murmur in the literal sense of the word This term, however, has been universally adopted to describe these acoustic phenomena and includes most abnormal sounds produced within the heart and great vesels Pericardial friction sounds are not considered murmurs.

Many murmurs are indicative of valvular or other important defects yet *others are unimportant and do not necessarily imply the presence of a heart lesion Conversely, severe heart disease often exists in the absence of murmurs*

Furthermore, in many instances a correct diagnosis cannot be made on the basis of murmurs alone Changes in the size of the heart chambers and character of heart sounds, the presence and location of thrills, and changes in the pulmonary and peripheral circulations are matters of equal, often of greater, importance One must often utilize facts obtainable by history, inspection, palpation, percussion, blood pressure determinations, data concerning the status of the lungs and peripheral circulation, x-ray, and electrocardiography in order to establish a diagnosis

Production

Murmurs in the heart or great vessels are thought to be due to eddies formed in the blood stream, presumably produced by its passage from a heart chamber or channel of lesser caliber into one of greater caliber, with resultant disturbance of the normal streamline flow Roughening of the endocardial surface by disease may be a contributing factor

The greater the pressure differential and the difference in caliber between the two channels and the more rapid the blood flow, the more intense will be the murmur If the change in caliber from the lesser to the greater chamber is sudden, as, for example, with rigid heart valves, there is greater eddy formation and consequently a louder murmur than if the change in caliber is more gradual, as in the case of blood passing from the left ventricle into a slightly dilated aorta Other factors modifying the intensity and quality of the murmur are the object of the impact of the jet of blood, direction of blood flow, form of the valve affected, viscosity of the blood, and presence of factors in the chest and its wall which affect transmission of sound

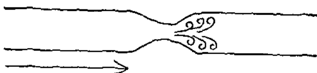


FIG 12 3 Schematic concept of murmur production by eddy formation in stenosis of a valve Arrow shows direction of blood flow Curved lines indicate eddies created by passage of blood from narrowed orifice into wider chamber

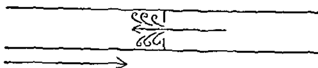


FIG 12 4 Schematic concept of murmur production in regurgitation through a valve Lower arrow shows normal direction of blood flow, upper arrow, reverse flow through incompletely closed valve Curved lines indicate eddies

With rare exception, all important cardiac murmurs are produced at or near a valve orifice by one of the following

Structural Disease of a Valve. A pathologic process may stiffen, shorten or otherwise deform the leaflets so that complete closure does not take place (*insufficiency* or *regurgitation*). Depending on the valve involved, the blood, during ventricular systole, flows backward from a contracting ventricle to a relaxing auricle (*mitral* or *tricuspid regurgitation*), or, during diastole, from the aorta or pulmonary artery to a relaxing ventricle (*aortic* or *pulmonic regurgitation*)

Comparable pathologic changes may prevent the leaflets from opening properly or narrow the normal orifice by scar tissue (*stenosis*) Failure of the mitral orifice to permit proper flow of blood during diastole from left auricle into left ventricle is *mitral stenosis*, narrowing of the aortic orifice, hindering egress of blood during systole from the left ventricle into the aorta is *aortic stenosis*. Lesions of the tricuspid and pulmonic valves, similarly produced and named, are comparatively rare Deformity of a valve sufficient to produce appreciable stenosis will also prevent proper closure, so that with stenosis some degree of insufficiency is almost always present, although it may not be apparent clinically.

Widening of a Valvular Orifice. If a chamber of the heart becomes dilated, the leaflets of one of its valves may fail to close the orifice completely and *relative* insufficiency results. Or, if the chamber distal to an orifice dilates and the chamber proximal to it remains normal, the valve orifice will be relatively narrow in comparison to the dilated chamber and *relative* stenosis will be the result.

Dilatation of Ascending Aorta or Pulmonary Artery. The blood flows through a normal orifice into a vessel whose caliber is wider than normal

Abnormal Communication between Heart Chambers or Vessels. Flow of blood through an abnormal channel such as a congenitally patent interventricular septum or patent ductus arteriosus will produce a murmur.

Endocardial Vegetation. In bacterial endocarditis a murmur may be caused by the presence in the blood stream of a large vegetation; one end of the tissue is attached to the ventricular wall or a valve leaflet while the other floats free in the stream. This is by no means as common as the causes noted above.

Important Diagnostic Features

In estimating the importance of a murmur, all of the foregoing must be kept in mind, especially the fact that murmurs must be interpreted in the light of additional findings—cardiovascular and others—made in the case. The important facts to be established about the murmur itself are:

1. Place in the cardiac cycle.
2. Area of distribution.
3. Quality.
4. Intensity.
5. Relation to exertion, respiration, and position of the patient.

Place in the Cardiac Cycle. Systolic Murmur. The first and most important fact to establish about a murmur is its time relation to the heart sounds. A systolic murmur is one which coincides with the first sound, totally or partially masking it, or occurs in any part of the period between the first and second sounds. One which does not begin until the first sound is ended is termed a *late* systolic murmur.

Diastolic Murmur. This occurs at the time of the second sound, partially or totally masking it, or follows the second sound, occurring between it and the next first sound. A murmur masking or occurring immediately after the second sound is known as *early* diastolic, one starting in the middle of diastole as *mid*-diastolic, one appearing toward the end of diastole, as *late* diastolic. The term



Fig. 12.5. Schematic representation of a systolic murmur following first sound. In this diagram and those which appear subsequently, heart sounds are represented by blocks, murmurs, by vertical lines.



Fig. 12.6. Short, early, diastolic murmur following second sound.



Fig. 12.7. Crescendo diastolic murmur beginning in mid diastole and ending at start of succeeding first sound.

presystolic is frequently applied to the murmur which begins in the middle or toward the end of diastole and continues up to the first sound of the following cycle If a *murmur replaces or masks a* cardiac sound it is often, but not necessarily, more serious than one which accompanies but does not replace the sound.

A *systolic murmur*, particularly one which appears late in systole, does not necessarily indicate valve disease *With one or two exceptions, diastolic murmurs always indicate valve disease* and, generally speaking, are of graver significance than many systolic murmurs A short mid-diastolic murmur, for example, may be the most important sign of mitral stenosis, and a faint early diastolic murmur may be the earliest recognizable indication of syphilitic aortitis

A long murmur does not necessarily indicate more severe disease than a short one. In fact, the latter, particularly if diastolic, may indicate serious heart disease, while some longer murmurs, especially if systolic, may have quite the opposite significance.

One of the commonest errors made in the diagnosis of cardiac disease is that of timing murmurs incorrectly Because of the difference in the significance of systolic and diastolic murmurs, this may have grave consequences. Familiarity with the character and rhythm of the heart sounds enables the experienced observer to time a murmur Occasionally, masking of sounds, rapid heart rate or abnormal rhythm makes this difficult Where there is any doubt, the cardiac systole should be identified by watching or feeling for its impulse at the apex or over a carotid artery. The radial pulse cannot be used, because of the time it takes the pulse wave to reach the periphery

Area of Distribution. To localize a murmur is to find its point of maximum intensity Murmurs heard loudest in the region of the apex impulse—whether in its normal position or displaced—are, in the majority of cases, produced in the mitral region Occasionally a mitral murmur is best heard midway between the normal position of the cardiac impulse and the ensiform cartilage or, rarely, a few centimeters above this site

Systolic murmurs heard most clearly in the second *left* intercostal space are usually produced at the pulmonic orifice or just above it in the conus pulmonalis Many congenital heart defects produce murmurs here. Murmurs heard best in the second *right* intercostal space are usually produced at the aortic orifice But in a large proportion of cases *aortic diastolic murmurs are more intense to the left of the sternum, usually in the region of the third or fourth left costal cartilage*, less often near the apex of the heart, in the axilla, or over the lower part of the sternum

A murmur is often audible over more than one valve area or over the entire precordium, and it then becomes a matter of conjecture as to whether one is dealing with a single valve lesion or with more than one This situation quite obviously arises only when such a murmur, audible at various points, occurs everywhere in the same part of the cardiac cycle, that is, is everywhere systolic

or everywhere diastolic The decision in such a case is often difficult, but, generally speaking, it can be made on the basis of the following points·

The murmur produced by each of the valvular lesions has its own characteristic area of distribution It is usually heard loudest over the valve area, and its intensity diminishes with increasing distance from that area. Any murmur whose distribution does not extend beyond one of these areas and whose intensity diminishes as the stethoscope is moved away from its assumed valvular area is probably due to one valve lesion

If, one the other hand, a murmur is heard which decreases in intensity as the stethoscope is moved further away from the valve area and seems to increase as the stethoscope approaches a second valve area, it is quite likely that murmurs are being produced at two valve orifices, rather than at one Furthermore, should the sound diminish as the stethoscope is again moved and increase as a third area is approached, it can be assumed that there are probably disturbances at three orifices Such an assumption would have an even sounder basis if one notes a change in pitch and quality of the murmur as the stethoscope is moved from one area to another Figure 16 27 represents the distribution of a systolic murmur in a case of mitral regurgitation and aortic stenosis One will hear over the lower precordium a systolic murmur, loudest at the apex If the stethoscope is moved toward the aortic area, this murmur will probably first become fainter, then gradually increase in intensity as the receiver nears the aortic area, reaching *a second maximum at this point Difference in pitch and quality*, best noted at the two points of maximum intensity will be expected In mitral insufficiency alone, the systolic murmur would probably fade out entirely as the aortic region was approached or at least become much less intense with increasing distance from the mitral area

Quality. The most common type of heart murmur has a blowing quality much like the sound of a breath forcibly exhaled through loosely pursed lips Blowing murmurs may be low-pitched, resembling the sound of air passing through a large tube, or high-pitched, more like a whistle This last type merges into that known as the musical murmur, in which there is a definite musical sound whose pitch can be identified Rasping sounds often characterize the louder varieties of murmurs Very rasping murmurs are associated with rigidity of the valve leaflets or with marked narrowing of the orifice

There is one type of sound which, though included under the general name murmur, differs from any of the other sounds just described This murmur begins in mid- or late diastole and seems to increase in intensity as the first sound of the next cycle is approached It has a rumbling, rolling sound, resembling a short drum-roll It is usually associated with well-developed stenosis of the mitral valve and is otherwise known as the diastolic *roll* or *rumble* Very rarely, in mitral stenosis, instead of the characteristic rumble one may hear a mid-diastolic blow

Murmurs may be accented at their end or beginning that is, they may be of the crescendo type, growing louder toward the end, or of the decrescendo type, with maximum intensity at the beginning

Intensity. The intensity of murmurs is variable, depending chiefly on factors already mentioned:

1. Degree of change between calibers of the passages through which the blood is flowing
2. Degree of pressure differential between the passages.
3. Speed of the blood flow.
4. Thickness of the tissues between the heart and surface of the chest

Murmurs may be so faint that they are audible only under the most favorable auscultatory circumstances. Rarely they are so loud that they can be heard at some distance from the chest. Between these two extremes there are all degrees of intensity. It is customary to classify murmurs on the basis of their loudness, *grade 1* indicating the faintest audible sound, and *grade 5*, the loudest.

From the diagnostic standpoint, the intensity of a murmur is usually less significant than its other characteristics. Occasionally it does give important evidence concerning the actual progress of a case under observation or the degree of change the disease has produced. A loud murmur may indicate severe disease, on the other hand, it may not. The same is true of a faint murmur. When a heart begins to fail, a murmur may become less intense, because pressure differential between chambers or speed of the blood flow has been decreased by weakening of the myocardium. With proper treatment, the myocardium may regain strength, blood flow and pressure differential may increase, and the murmur become louder. In this instance, increase in the intensity of the murmur is an indication of improvement. In mitral stenosis, for example, a relatively loud murmur caused by blood being forced through a narrow opening may become less intense as failure sets in and, with improvement, become more intense. On the other hand, if the myocardium is not seriously weakened, increase in the intensity of the murmur indicates increasing stenosis of the valve.

In a case of mitral insufficiency, a loud systolic murmur might indicate that the blood is being forced backward through a narrow opening and predicate very little regurgitation. But if as a result of further damage to the heart valve the degree of insufficiency becomes greater, the murmur might become less loud because of increase in the caliber of the orifice through which the leak is occurring. In this case decrease of the murmur is a bad sign.

In general, it may be said that the most important point about the intensity of a murmur is its increase or decrease while under observation, and not its loudness at any one time.

Relation to Exercise, Respiration, and Position. EXERCISE. Murmurs which are inaudible or just barely heard with the patient at rest may, because of the increased blood flow brought about by exercise, be more clearly heard after physical exertion of some kind, such as walking about briskly or climbing a flight of stairs. However, if the myocardium is in a greatly weakened condition, exercise may, by increasing the strain on the muscle, do just the opposite and still further lessen the intensity of a murmur.

RESPIRATION. Most murmurs are better heard with the lungs in the position of full expiration and become fainter during inspiration as the expanding lungs

cover the heart. This is particularly true of the diastolic murmurs of early aortic regurgitation and early mitral stenosis. On the other hand, the cardiopulmonary murmur is more apt to be heard best at some other point in the respiratory cycle.

Position. Almost all cardiac murmurs are affected by the position of the patient. Those which are almost inaudible while he is sitting or standing might be clearly heard when he lies down, and those not well heard in the dorsal position might be brought out by having the patient lie on his left side or face downward This is particularly true of the mid- or late diastolic murmur of mitral stenosis and of the early diastolic murmur of aortic regurgitation

Classification of Murmurs

Murmurs having no clinical significance are termed physiologic; those due to disease, pathologic

Physiologic Murmurs

These are almost always systolic in time.

Intracardiac or Intravascular. Due to causes not clearly understood, these have the following characteristics:

1. Usually confined to a limited region, the majority are heard over the pulmonic valve area, the others at the apex.
2. As a rule they are short, rarely occupying all of systole, and soft and blowing in quality.
3. They become louder as the lungs are deflated, and softer with inspiration.
4. They vary with position of the body. Generally, they are loudest, and sometimes only heard, in recumbency.
5. They are apt to be evanescent or inconstant, often appearing after hard exertion and disappearing within a few minutes or hours
6. They are not associated with cardiac enlargement or other evidence of heart disease

Extracardiac (Cardiopulmonary). Although the term cardiorespiratory is often used to describe these murmurs, cardiopulmonary is preferable, since it indicates that they actually arise in the lung Although often present in normal persons these murmurs are more likely to occur in the patient whose free lung margin is fixed by adhesions Squeezing out of air by cardiac action from a small portion of lung adjacent to the heart is thought to be the cause of those which are systolic, sucking of air into adjacent lung tissue, the cause of the rare ones which are diastolic Cardiopulmonary murmurs are in most respects similar to intracardiac or intravascular physiologic murmurs. The intracardiac or intravascular murmur, however, is usually best heard with the lungs at the point of expiration, whereas the cardiopulmonary murmur is often most clearly audible with the lungs partially inflated As the patient breathes slowly, this murmur [illegible]

[illegible] regions.

Pathologic Murmurs

These may be either systolic or diastolic and are caused by:

Structural Valvular Disease. Here the murmur is due to a defect of the valve, such as rheumatic mitral stenosis or syphilitic aortic insufficiency

Dilatation of a Heart Chamber or Great Vessel. This may be secondary to:

1 Cardiovascular disease, such as, (a) relative mitral insufficiency in a case of hypertension with left ventricular enlargement, or (b) dilatation of the aorta due to arteriosclerosis.

2. Disorders which secondarily affect the circulatory system, such as thyrotoxicosis, anemia, and deficiency states

Congenital Cardiovascular Defect. The murmur is caused by flow of blood through the abnormal channel.

Rupture of an Intracardiac Structure. A murmur may suddenly appear after perforation of a valve leaflet, which is most likely in subacute bacterial endocarditis. Following myocardial infarction or trauma, rupture of a papillary muscle or the interventricular septum will be attended by the appearance of a new murmur.

Floating Tissue in the Blood Stream. This phenomenon, which is relatively rare, is most often found in bacterial endocarditis

The interpretation of any murmur depends fully as much on other clinical features of the case as on the characteristics of the sound itself

Systolic Murmurs

At the Apex. The systolic murmur heard best at the apex is common Its importance is often difficult to estimate It may begin early in systole, partially or totally masking the first sound, or it may begin later, after the first sound has ceased. Almost always it is blowing in quality Its pitch and intensity vary The extent of its transmission depends upon the degree of intensity If sufficiently loud, this murmur may be heard in the axilla, in the middle of the left back, or over the upper part of the precordium; but in any case its point of maximum intensity is in the region of the apex As a general rule the louder and longer this murmur the more apt it is to be pathologic.

PHYSIOLOGIC The murmur may be intracardiac or cardiopulmonary. It usually occurs in early or mid-systole, has the characteristics described above, and is not associated with other evidence of heart disease

PATHOLOGIC. There are two causes

1 Insufficiency of mitral valve This may be brought about by actual disease of the valve, by left ventricular dilatation from any cause, or by a combination of the two The murmur is usually loud and harsh, occupies most of systole, often masking the first sound, may be transmitted to the axilla or back, and shows little or no variation with changes of position or with respiration.

2. Transmission of a systolic murmur arising elsewhere. An example is the loud murmur of aortic stenosis.

In the Pulmonic Area. PHYSIOLOGIC. This murmur is very common, especially in children and young persons, particularly those with thin chest walls It is probably due to sudden filling and distention of the pulmonary artery, which is near the surface at this point. It begins early in systole and may occupy all of systole, is blowing, sometimes harsh, may be limited to the pulmonic valve area or heard over the precordium, is louder at expiration, may diminish or disappear with change in position, and is often increased by exertion, fever or other cause of vigorous heart action

PATHOLOGIC. The most likely causes are:

1 Stenosis of pulmonic valve. This murmur is harsh, loud, occupies most of systole, may be heard widely over the left upper chest, and is usually accompanied by a palpable thrill. The pulmonary second sound may be accentuated or diminished.

2 Patency of ductus arteriosus.

3 Transmission. Sometimes a systolic murmur may be so clearly transmitted from the aortic area that it is impossible to tell whether it arises in the aortic, pulmonic or both areas

In the Aortic Area. Most systolic murmurs here are pathologic and due to:

1 Dilatation of aorta due to arteriosclerosis with or without hypertension, or syphilis This murmur is fairly well localized and blowing in quality; in severe cases it may be intense and harsh Occasionally it is transmitted to the vessels of the neck. It does not vary with change in position but may be better heard with the lungs deflated The aortic second sound is often increased.

2 Stenosis of aortic valve. Characteristically, this murmur starts early in systole, frequently masking the first sound at the base, and shows transmission into the vessels of the neck and toward the apex It is loud and rough, often accompanied by a palpable, systolic thrill, and often followed by the diastolic murmur of aortic regurgitation. The second sound may be diminished or absent because of failure of the valve leaflets to snap shut in the normal manner.

3 Aneurysm of aorta The quality of the murmur is variable.

4 Transmission of a systolic murmur originating elsewhere

In Other Cardiac Areas. A systolic murmur heard best to the left of the sternum at the level of the third to fifth intercostal spaces is most likely due to interventricular septal defect. It is loud, harsh, often accompanied by a thrill and may extend upward along the sternal margin. When heard over the lower sternum, a systolic murmur is usually transmitted from elsewhere; rarely it is due to tricuspid regurgitation

DIASTOLIC MURMURS

Except for the very rare diastolic cardiopulmonary murmur, diastolic murmurs are always pathologic and point to some important disorder, as indicated below

At the Apex.

1. Structural stenosis of mitral valve The murmur begins in mid-diastole

and varies from a short, barely audible, rumbling sound to a loud, rough, rolling or rumbling murmur occupying the second half of diastole and followed by a snapping first sound Rarely it is a blow. The murmur is usually localized It may be more clearly heard with the bell-shaped than with the Bowles chest piece When inaudible with the patient in the upright or dorsal position, a change to the left-lateral will often bring it out This murmur may be loudest at onset, but sometimes will reach its crescendo at the very end, just before the sharp first sound is heard A palpable precordial thrill frequently accompanies it

2. Relative stenosis of mitral valve. In cases of free aortic regurgitation and sometimes with pronounced left ventricular dilatation from other cause, a murmur is produced by blood passing through a mitral orifice which, as compared with the dilated left ventricle, is relatively narrowed Known as the *Austin Flint* murmur, it has the same characteristics as that of structural mitral stenosis, but is usually not as pronounced.

3. Transmission. A loud, blowing diastolic murmur due to aortic regurgitation may be transmitted downward to the apex. It will be louder in the aortic region or along the left border of the sternum.

In the Pulmonic Area. Relative insufficiency of pulmonic valve Dilatation of the pulmonary artery and valve ring secondary to increased pulmonary vascular tension may produce a murmur similar to that of mild aortic regurgitation, from which it is often indistinguishable without the aid of other clinical findings. It is rarely as intense as the murmur of aortic insufficiency, and in contrast to the latter is likely to be heard best at the second left interspace, instead of further down The pulmonary second sound is increased The most common cause is mitral stenosis, here the murmur is known as the *Graham Steelle* murmur.

In the Aortic Area.

1 Structural insufficiency of aortic valve—rheumatic, syphilitic or calcareous. This murmur is blowing and begins early in diastole immediately following the second sound In severe cases, it occurs simultaneously with the second sound, partially or totally masking it If the valve leaflets are firmly fixed by disease, the second sound will be entirely absent The murmur may occupy only a short period in diastole or, if intense, all of it. *When not pronounced it is most often best heard or only heard just to the left of the sternum at the level of the third or fourth intercostal space* Rarely, it will be more clearly audible at the aortic valve area or at the lower end of the sternum It is better heard with the Bowles than with the bell-shaped chest piece Sometimes it is so soft that it can be detected only when the patient's lungs are at the point of full expiration and even then only with the patient in one particular position, usually sitting or standing with trunk bent well downward When loud, it can be easily heard in the aortic area and along the left sternal border.

The discovery of this murmur in its early stages, when it is a short, blowing sound in early diastole, is often of great importance, particularly in the early

diagnosis of syphilis of the aorta or rheumatic disease of the aortic valve. In appropriate cases it should be searched for with the patient in various positions and with his lungs deflated.

2. Relative insufficiency of aortic valve. This can occur as a result of: (a) aortic dilatation from arteriosclerosis or syphilis, without actual damage to the valve, (b) marked hypertension, (c) thyrotoxicosis; (d) severe anemia. The murmur has in general the same character in all four disorders but in the first two is not as loud, intense or long as in structural valve disease

3 Transmission A diastolic murmur originating elsewhere is sometimes transmitted to the aortic area but is rarely confused with a true aortic murmur.

In the Tricuspid Area. Stenosis of tricuspid valve The murmur is similar to that of mitral stenosis except for the fact that it is found in the region of the lower end of the sternum, not at the apex Diagnosis cannot be made on the basis of auscultation alone, the presence of other signs is necessary

All diastolic murmurs merit careful investigation, for even if not due to actual valve disease, they almost always indicate the presence of some serious disturbance

Metamorphosing Murmurs

Change of murmurs resulting from variation in strength of heart beats has already been described

In acute or subacute bacterial endocarditis, if a vegetation is forming or changing shape on a valve or if a leaflet ruptures, a new murmur may appear or an existing murmur may show a change in character Following myocardial infarction or trauma, rupture of a papillary muscle or the interventricular septum will create a comparable modification When the mitral valve or interventricular septum is affected, the murmur is systolic, when the aortic valve is the seat of the trouble, diastolic

Continuous Murmurs

These murmurs, although they may be audible over the heart, are almost always of vascular origin

Venous Hum. This is heard above the clavicle in children, especially on the right, and may extend downward over the upper sternum where it can be mistaken for a heart murmur It is usually faint, low-pitched and continuous, rarely loud and roaring It can be increased on either side by having the patient twist his head in the opposite direction so that the tissues are stretched It can be differentiated from a heart murmur by pressing one's finger over the jugular vein so as to stop blood flow in the vein This will cause the hum to disappear It has no pathologic significance

Patent Ductus Arteriosus. This congenital anomaly produces a murmur heard most clearly in the first or second interspace to the left of the sternum, but somewhat lateral to the pulmonic valve area It may be intense or quite faint, it continues throughout the cardiac cycle but with systolic accentuation, and it has

a humming or "machinery" quality. Occasionally no diastolic phase is heard. When the murmur is intense a thrill may be palpable.

MURMURS OVER PERIPHERAL VESSELS

Systolic. The systolic murmur, due to dilatation of the aorta or aortic stenosis, will, if sufficiently intense, be transmitted to the vessels of the neck.

Local narrowing of the lumen of any of the larger arteries by such causes as an inflated blood pressure cuff, cicatricial tissue, or tumor will produce a murmur synchronous with the pulse wave

Arterial systolic murmurs may also be audible over: (1) the gravid uterus; (2) the anterior fontanel in infants, (3) the thyroid gland in many cases of thyrotoxicosis. In the last, one may hear a bruit which is generally continuous, but louder during systole It may be substernal.

Diastolic. A diastolic arterial murmur may be heard over the larger arteries in pronounced aortic regurgitation It may be intensified by exertion or by slight pressure over the vessel. In the latter event it is best heard *proximal* to the point of pressure, because, as a result of the low diastolic pressure, the blood flow through the constricted area is reversed during diastole. This is known as *Duroziez's sign* In peripheral vascular dilatation from such causes as hyperthyroidism and marked fever, a diastolic murmur may be heard in the peripheral arteries if pressure is applied. But the murmur is heard *distal* to the point of pressure, due to the rapid forward flow of blood into the dilated vascular bed beyond.

Continuous. Arteriovenous fistula, a direct connection between a large artery and vein, gives rise to a continuous murmur Trauma is the usual cause The murmur has a humming quality, is usually increased during systole, and may be accompanied by a palpable thrill.

PERICARDIAL FRICTION SOUND

This is comparable to the sound produced by pleural friction Under normal circumstances, the pericardial surfaces are so smooth that sounds are rarely produced by their contact. However, faint scratchy sounds—systolic, diastolic or both—are occasionally heard along the left border of the sternum in dehydrated patients, and when heart action is increased, as by exercise or thyrotoxicosis They are presumably caused by rubbing together of the normal pericardial surfaces by the vigorous movement of the heart or pulmonary artery and are not an indication of pericardial inflammation

When the pericardial surfaces are inflamed, friction is produced by the roughened areas rubbing against each other during the cardiac cycle The sounds vary in intensity from soft and almost blowing to rough, grating or shuffling. They may occur during the greater part of each cycle with accentuation in systole, or be heard only in systole When not intense they may be easily confused with blowing murmurs, if loud they may conceal murmurs or hamper their interpretation. Friction is most commonly audible just to the left of the sternum at the level of the third or fourth interspace but may be heard in a

limited area anywhere over the precordium or over the entire region It is likely to be found if the patient lies on the right side, and intensified if, with his lungs deflated, he leans forward and to the left, thus bringing his heart closer to the chest wall In contrast to murmurs, the sounds change from day to day, an important point in distinguishing between the two Pressure on the chest wall with the stethoscope may increase the sounds so that they seem nearer the ear. Pronounced friction may be palpable

Sounds easily confused with pericardial friction may be heard in cases of acute pleuritis in which inflamed pleural surfaces close to the heart are rubbed together by cardiac movement (*see* Chap 23).

13

THE ELECTROCARDIOGRAM

Although not a part of physical examination, electrocardiography is so important in the diagnosis of many forms of heart disease that a description of the procedure is included here to provide some understanding of the electrocardiographic variants discussed in subsequent chapters Without benefit of this procedure, proper diagnosis and intelligent treatment of certain cardiac disorders is often impossible

An electrocardiogram is a graphic record, registered by a galvanometer, of the small variations of electric potential associated with myocardial contraction Since the electric currents set up in the myocardium spread to the periphery, they can be picked up by electrodes placed *anywhere* on the body surface and conveyed to the galvanometer by connecting wires The extremities and chest wall are the regions used. When the heart is at rest the record shows a straight horizontal line Currents developing with myocardial activity appear as upward (*positive*) or downward (*negative*) deflections of this line

The machine is so designed that, (1) other currents set up in the skin or elsewhere in the body are neutralized; (2) a change of potential of 1 mv. can be expressed as a deflection of 10 mm , thus permitting standardization and consequent accurate comparison of different tracings; and (3) time intervals of 0 04 second are recorded by vertical lines, 1 mm apart.

Bipolar Limb Leads

Known also as the *standard* leads, these are the ones which were first used in clinical electrocardiography The electrodes are placed *on the extremities*; a closed circuit is obtained by use of a control switch so designed that *only two* electrodes are connected with the galvanometer at the same time The following combinations, effected by turning the switch to the proper positions, are used:

1 Lead 1. Right arm and left arm
2. Lead 2. Right arm and left leg
3 Lead 3. Left arm and left leg

The electrodes in lead 1 are arbitrarily connected to the galvanometer so that the left arm electrode is always the positive (*exploring*) and the right arm the negative (*indifferent*) electrode. In lead 2 the left leg electrode is positive and the right arm negative, in lead 3 the left leg electrode positive and the left

arm negative. Any accidental reversal of lead wires will produce a distorted set of tracings

Although the appropriate electrodes are placed on the right and left wrists and just above the left ankle, the limbs function merely as wires leading from the vicinity of the shoulders and symphysis pubis which, for all practical pur-

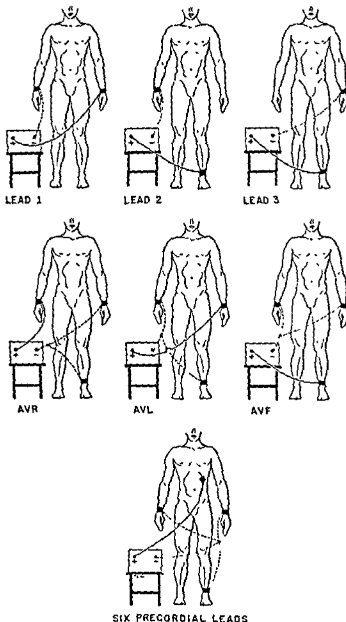


FIG 13 1 Electrode positions for conventional electrocardiogram.

poses, are equidistant from the source of the electrical potential (the heart) Any deflection in lead 2 is equal to the algebraic sum of its corresponding deflections in leads 1 and 3

Bipolar Precordial Leads

The exploring (positive) electrode is placed over the precordium and one of the extremity leads is used to complete the circuit The precordial electrode is designated C, the indifferent electrode R, L or F, depending on whether it is placed on the right arm, left arm or left leg. The letter F (foot) is used for the left leg to avoid confusion with the L of left arm.

The various positions at which the exploring electrode is placed have been arbitrarily indicated by numbers as follows.

1. Fourth intercostal space just to the right of sternum

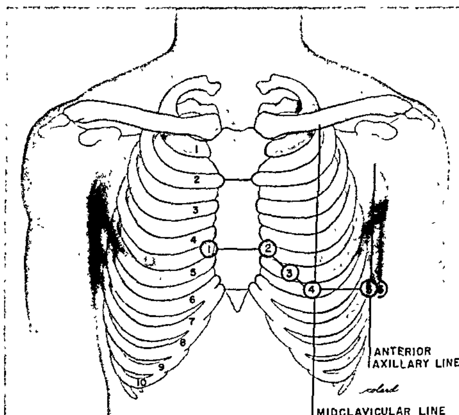


Fig 13 2 Positions of exploring electrode for conventional precordial leads

2. Fourth intercostal space just to the left of sternum.
3. Midway between positions 2 and 4.
4. Fifth intercostal space in midclavicular line
5. Directly lateral to position 4 in anterior axillary line.
6. Directly lateral to position 5 in midaxillary line.

Thus lead CF1 indicates that the indifferent electrode is on the left leg and the exploring electrode is in position number 1 on the chest, lead CR3, that the indifferent electrode is on the right arm and the exploring electrode at number 3 chest position At the Massachusetts General Hospital and many other clinics, bipolar precordial leads have, in recent years, been replaced by unipolar chest leads and are no longer routinely used. Consequently, the former are not discussed in the following pages.

Unipolar Limb Leads

In contrast to the bipolar leads which record differences in potential between two electrodes at a given moment, each unipolar lead registers the actual change of potential occurring at the *point of contact of the exploring electrode* This can be accomplished by placing indifferent electrodes on three extremities and leading them into one "central terminal"; their combined potential will always equal zero If a fourth electrode (exploring) is applied in turn to each of the three limbs, the current brought in through this will, in each case, closely reflect the changes of potential occurring at the point of contact These are the unipolar limb leads (VR, VL and VF) However, because of their low voltages and consequent small waves they are not practicable for clinical use. Greater potential variations and hence more easily readable tracings are obtained by the use of only three electrodes (*augmented unipolar limb leads*). The lead-offs from two limbs are joined to form the indifferent electrode while the third limb is used for the exploring electrode This method is now universally employed Machines of recent design are so wired that with an electrode on each arm and on the left leg one can, by use of a selector switch, obtain in turn the three augmented unipolar limb tracings. These combinations are designated as follows

Lead AVR Exploring electrode, right arm, indifferent electrode, left arm and left leg

Lead AVL Exploring electrode, left arm, indifferent electrode, right arm and left leg

Lead AVF Exploring electrode, left leg; indifferent electrode, right arm and left arm

The amplitudes of the augmented unipolar limb lead complexes are approximately 15 per cent smaller than those of the bipolar limb leads The algebraic sum of any three corresponding complexes equals zero The chief value of the augmented limb leads is in determining, together with the precordial leads, the electrical position of the heart with respect to all three of its axes and also at times to assist in the diagnosis of posterior myocardial infarction However, for the trained observer one group of limb leads along with the

precordial leads will usually suffice; with experience, he can quite accurately predict from the appearance of the bipolar limb leads, the shape, size and direction of the complexes in the unipolar limb leads, and *vice versa.*

Unipolar Precordial Leads

All three limb leads are combined to form the indifferent electrode; the exploring electrode is placed in turn at the six positions used for the bipolar chest leads. These unipolar leads are designated V1–V6. As noted above, they have almost entirely replaced the bipolar chest leads in routine examinations They have proved to be the most valuable in the diagnosis of certain cardiac disorders, especially myocardial infarction, ventricular hypertrophy and strain, and bundle branch block.

In this and following chapters we will not always discuss all leads but only those which serve to illustrate pertinent points

THE NORMAL ELECTROCARDIOGRAM

The electrocardiogram of the normal cardiac cycle consists of a series of deflections which have been arbitrarily named P wave, QRS complex, and T wave Following the T wave, a small unimportant deflection (U wave) is present in some tracings. When tracings are described, each deflection is indicated by its appropriate letter, followed by the designation of the lead through which it was obtained. For example, P1 indicates the P wave in lead 1; QRS AVL, the QRS complex in the augmented unipolar limb lead taken with the exploring electrode on the left arm; T V5, the T wave in the unipolar precordial lead taken with the exploring electrode in position 5 In surveying a tracing, it is customary first to determine rate, rhythm, and duration of P-R, QRS and Q-T intervals and then to study in turn the individual waves and complexes (P, QRS and ST-T) as they appear in successive leads

The records of corresponding leads taken from different persons with normal hearts conform to fairly similar patterns but vary somewhat because of such factors as body build and heart position

Each deflection—P, QRS, and T—has its individual configuration, in the same person corresponding deflections in the various leads conform to the same pattern but are never exactly alike In the case of the normal heart, their amplitude and direction (upward or downward) are dependent on the lead and heart position. An upward deflection is measured from the top of the baseline to the peak of the wave, a downward deflection from the bottom of the baseline to the nadir of the wave As noted above, in the bipolar limb leads the algebraic sum of the heights of the corresponding waves in leads 1 and 3 equals the height of the same wave in lead 2. In the unipolar limb leads the algebraic sum of the three corresponding waves equals zero

Although the normal unipolar precordial pattern going from position 1 to position 6 (V1–V6) varies somewhat from person to person, it generally shows

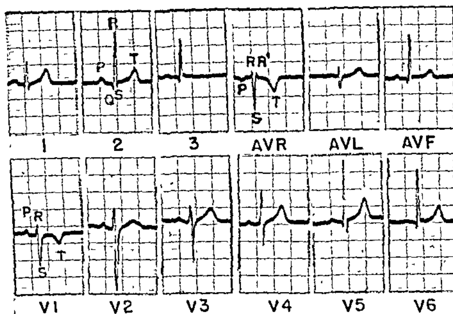


FIG 13 3 Normal electrocardiogram In this and subsequent tracings standardization is uniform the galvanometer was always adjusted so that introduction of 1 millivolt into the circuit would create a deflection of 10 millimeters Fine horizontal lines are 1 millimeter apart Spaces between heavy vertical lines represent 0 20 second, between fine lines, 0 04 second In the following chapters when a variant is adequately demonstrated by a single lead, only the one which shows it most clearly has been reproduced

a fairly constant and characteristic sequence of changes Chiefly because of dissimilarity of heart position, the deflections of the six unipolar precordial leads in one normal person may be somewhat different from their counterparts in another But if the six V leads in the first person are viewed as a series and compared to the six in the second viewed as a series, the two patterns and the changes which occur from position to position (V1, V2, V3, etc) will be found to be quite similar The same applies to the bipolar chest leads Here the sequence is almost identical with the unipolar leads as far as the QRS complexes are concerned, but differs somewhat in the case of the T waves.

The range of normal is wide Correct interpretation of the electrocardiogram requires experience because of variations due to such factors as postion of the subject, his age, degree of recent exertion, emotional status, position of heart, thickness of thoracic wall, and presence or absence of some non-cardiac disturbances such as emphysema or pleural effusion *An electrocardiogram which does not conform absolutely to the usual picture does not necessarily indicate heart disease Nor does a normal electrocardiogram exclude heart disease* Interpretation of any tracing is most dependable when one is familiar with the pertinent clinical features of the case. Furthermore, *it is often highly important* to compare a patient's electrocardiogram with any taken previously, changes in the pattern are always of significance.

The P Wave

This represents the electrical phenomena which precede and initiate *auricular* contraction. It consists of a curve—rather blunt, averaging 1–2.5 mm in amplitude in the limb leads and rarely over 0.1 second in duration. It is normally upright in leads 1 and 2 but may be upright, diphasic or inverted in lead 3. It is usually largest in lead 2. The P wave in AVR, as well as the QRS and T complexes, is normally inverted. The P waves of precordial leads V1 and V2 are normally diphasic and become upright though smaller as V5 and V6 are reached.

The P-R Interval

This interval is measured from beginning of the P wave to beginning of the QRS complex. It represents *auriculoventricular conduction time*, that is, the time consumed by the wave of excitation in passing from its starting point, the sinoauricular node, through the auricles and the auriculoventricular node. It varies in length from 0.11 to 0.21 second, averaging 0.16 second and tending to be shorter with rapid, longer with slower, rates.

The QRS Complex

The QRS complex is the manifestation of electrical depolarization which precedes and initiates ventricular contraction. It comprises a group of rapid, sharp deflections normally totaling 0.05–0.1 second. An initial downward deflection is defined as a Q wave, an upward deflection as an R wave, a downward deflection following an R wave as an S wave. If, as sometimes happens, there are two upward deflections, the one following the S wave is designated R′.

The QRS deflections show marked variation in amplitude in different leads in the same person as well as in corresponding leads in different normal persons. In lead 2 the R wave varies usually from 10–20 mm and is lower in leads 1 and 3. Small, narrow Q waves may be seen in any of the limb leads, especially lead 3. S waves are narrow and of variable size, depending on position of the heart. As a rule in at least one of the bipolar limb leads the total excursion (arithmetical sum of the deflections above and below the baseline) of the QRS complex is greater than 5 mm; otherwise abnormally low voltage is present. A total deflection of 20 mm or more in one of the limb leads represents abnormally high voltage.

As previously stated, deflections of the augmented unipolar limb leads tend to be about 15 per cent smaller than those of the bipolar limb leads. In AVR, the QRS complex normally is either a deep Q followed by an R, or a completely negative deflection (QS). The direction taken in AVL and AVF depends to a large extent on the anatomic position of the heart with respect to its anteroposterior axis. In a *vertical* heart (right axis deviation by bipolar limb leads) the main QRS deflection is downward in AVL, upright in AVF; in a *horizontal* heart (left axis deviation by bipolar limb leads), the reverse is true. There are all gradations between these two extremes.

Because of closer proximity of the electrode to the heart in the precordial leads, the amplitude of QRS tends to be greater than in the limb leads. No acceptable standards of normal have yet been defined. The six precordial QRS complexes taken by the bipolar method will be almost identical in size and configuration to the corresponding QRS complexes taken by the unipolar method. Consequently, only the unipolar leads will be described. In V1, Q wave is absent, the complex consists of a small, narrow R wave followed by a deep, wide S wave. This pattern reflects the electrical changes over the right ventricle. In V6, the complex is usually a small, narrow Q wave followed by a large, wide R wave and, as a rule, no S wave. This represents the changes over the left ventricle. As one explores from V1 to V6, R wave tends to become taller and wider, S wave shorter and narrower, at approximately V3 (*transition point*) R wave and S wave will be nearly equal.

Notching or thickening (*slurring*) of one or both limbs of QRS is often observed in one or more leads, neither is of significance if the complex is of normal width.

Heart rate is determined by counting the QRS complexes in a given time interval. On most records the space between vertical lines is 1 mm. and represents 0.04 second. Every fifth line is heavy so that the space between two consecutive heavy lines represents one-fifth (0.2) second and 30 such spaces (15 cm.) represent 6 seconds. Thus, counting the number of QRS complexes on 15 cm. of tracing and multiplying by 10 gives the rate per minute.

The T Wave

The T wave represents electrical repolarization of the ventricles and appears near the end of mechanical systole. Its amplitude is variable, usually appreciably less than that of the accompanying QRS complex. In the limb leads it averages 1–4 mm., the range of normal in the precordial leads is wider. It is a much slower wave than the QRS deflection, averaging 0.1–0.25 second. It is upright in lead 1, almost always upright in lead 2 and may be *upright*, *flat* (*isoelectric*), downward or diphasic in lead 3. In the unipolar limb leads, T is downward in AVR, can be upright, flat or downward in AVL and is upright in AVF. In the unipolar precordial leads it may be upright, isoelectric or downward in V1 and V2 but is almost always upright in V3–V6.

Many factors not related to heart disease can alter the size and configuration of T waves and must be taken into consideration when their variations are being evaluated.

The Q-T Interval

Extending from the onset of QRS to the end of T, this measures the *total duration of ventricular systole*. It varies from 0.26 to 0.49 second, being shorter with rapid heart rates and longer with slow rates.

The S-T Segment

The interval between end of QRS and beginning of T is marked by a straight line on or barely above or below the baseline level. For reasons indicated be-

low, this segment and the T wave are usually described together (ST-T complex).

The U Wave

This is a small deflection which usually takes the same direction as the preceding T wave In some cases it is not evident; in others it may be seen in the *precordial leads*, less often in limb leads.

ABNORMALITIES OF ELECTROCARDIOGRAPHIC WAVES

Although abnormal electrocardiograms will be discussed in connection with their associated cardiac disturbances, the *common variants of the individual* complexes and the disorders in which they are usually found are indicated below

The P Wave

Increased Amplitude. In the limb leads, a P wave greater than 2 5 mm in height is abnormally tall (*cor pulmonale type*) One which is greater than 0 10 second in duration is abnormally broad and often macroscopically notched (*mitral type*) Either indicates auricular enlargement

Decreased Amplitude. *This is found in association with the decreased amplitude* of QRS discussed below It also occurs in severe hyperpotassemia

Absence. *In auricular fibrillation and flutter the P waves are replaced* by rapidly recurring deflections of the baseline (F waves) In paroxysmal tachycardia, premature beats, auriculoventricular block and auriculoventricular nodal rhythm, the P waves may be buried in the QRS complexes or T waves

Reversal. Normally upright P waves or normally negative P waves are often but not necessarily reversed in auricular premature beats, auricular paroxysmal tachycardia, auriculoventricular nodal rhythm (*retrograde P waves*), and, rarely, following ventricular premature beats In congenital dextrocardia with situs inversus, P1 is inverted along with reversal of the other complexes in lead I (*mirror image*) Lead AVL *gives the picture of the normal* AVR and *vice versa*

The P-R Interval

Prolongation. At a heart rate below 70, a P-R interval as long as 0 21 second is regarded as normal, at a rate of 130, 0 16 second is regarded as the upper limit of normal If the interval at a given heart rate exceeds the limit of normal, auriculoventricular block is present The delay may be so slight as to produce no change in heart rhythm (*delayed conduction time* or *first degree heart block*) It may be sufficient to result in an occasional dropped ventricular response at regular or irregular intervals (*second degree heart block*) or total failure of ventricular response (*complete heart block*) In the latter, the auricles contract at their own rhythm and the ventricles independently at a slow rate usually between 30 and 50 per minute

Shortening. In auriculoventricular nodal rhythm the P wave falls just before, within, or after the QRS complex. The P wave and QRS complex may

happen to fall close together in complete heart block and in some case of premature beats; this gives the appearance of a short P-R interval but actually no P-R interval exists since the impulses initiating auricular and ventricular contractions are independent of each other. In *Wolff-Parkinson-White* syndrome the P-R interval is 0 10 second or less but the QRS is of correspondingly longer duration so that the interval between the beginning of P wave and termination of QRS is within the normal range.

The QRS Complex

Axis Deviation. Although axis deviation is relatively unimportant in the interpretation of tracings, one must have the following facts in mind

LEFT axis deviation (horizontal heart) shows the following picture:

S3 greater than R3.

R1 greater than R2 or R3

Main QRS deflection upright in AVL, downward in AVF.

It is found in any disturbance such as high diaphragm or right pleural effusion which displaces the heart toward the left, and in disorders causing enlargement of the left ventricle, such as aortic insufficiency and hypertension

RIGHT axis deviation (vertical heart) shows the following·

S1 greater than R1.

R3 greater than R1 or R2

Main QRS deflection downward in AVL, upright in AVF.

This is a normal finding in children and in persons with ptotic builds It is also encountered when there is an abnormal load on the right ventricle as in mitral or pulmonic stenosis, certain congenital and cardiovascular defects and some cases of chronic fibropulmonary disease. In pulmonary emphysema without heart disease, it may be brought about by the shift of the heart downward and toward the right as a result of the low position of the diaphragm.

Increased Amplitude. In general, if the arithmetical sum of the R and S deflections in any of the limb leads is greater than 20 mm. it is regarded as increased This variant may be found in any disturbance causing left ventricular enlargement or strain It also occurs in ventricular premature beats It may be [illegible] here an abnormal pre-

[illegible] R and S deflections in none of the limb leads exceeds 5 mm , low voltage is present The same is true in the precordial leads if none of the sums exceeds 18–20 mm. Decreased amplitude is most likely to be found in one of the following disturbances:

1. Hypothyroidism
2. Severe myocardial disease, especially that due to coronary disease.
3. Large pericardial effusion or constrictive pericarditis
4. Extensive anasarca
5. Terminal or severe toxic states
6. Deficiency states
7. Emphysema The change is most likely to be found in the precordial leads

Notching or Slurring. If either occurs in one or two leads near the baseline it is of no significance. Otherwise it is seen in QRS prolongation.

Prolongation. Duration of the QRS complex greater than 0.10 second represents increased intraventricular conduction time and is found in:

1. Intraventricular block and bundle branch block.
2. Ventricular premature beats or ventricular paroxysmal tachycardia
3. Drug effects, as during administration of quinidine or procaine amide hydrochloride
4. Certain electrolyte disturbances

The ST-T Complex

Since disorders which create changes in the ST segments almost always cause changes in the T waves and *vice versa*, the two are discussed as a single complex. *In studying the ST segment one looks for elevation, depression, and configuration*—that is, whether the segment is flat, curved upward (*upward curving*), or *curved downward* (*sagging*). *Notable T wave variants are abnormally high or* low amplitude, reversed direction (usually inversion, since T waves in most leads are normally upright) and broadening (increased duration) The common causes of ST-T complex changes are

1. Coronary artery disease, including angina pectoris and coronary insufficiency (often only during an attack), and myocardial infarction
2. Ventricular strain
3. Bundle branch block.
4. Ventricular premature beats
5. Pericarditis
6. Myocarditis.
7. Myxedema
8. Deficiency states, perhaps
9. Electrolyte imbalance, especially hyper- or hypokalemia and hypocalcemia.
10. Effect of drugs, especially digitalis and quinidine
11. Tachycardia. When the tachycardia is not due to actual heart disease, the ST-T changes are usually slight and of no significance.

The Q-T Interval

Prolongation of this interval is seen in bundle branch block, certain electrolyte disturbances, and often following myocardial infarction Shortening is not uncommon in patients receiving digitalis therapy

The U Wave

Ordinarily, this is of little clinical significance In hypokalemia it may be prominent, especially in the precordial leads.

In reporting an *electrocardiogram, it is customary to record* rate, rhythm, duration of P-R, QRS and Q-T intervals, status of the various waves, changes evident since previous tracings, and the examiner's interpretation of his findings

(Waves or complexes within the normal range are not necessarily mentioned.) For example, a typical report might read as follows.

Rate. 75

Rhythm: Normal

P-R Interval 0.12 sec; QRS. 0.10 sec; Q-T: 0.38 sec.

P Waves: Normal

QRS Complexes Q waves prominent in leads 2, 3, AVF

ST Segments Slightly elevated in leads 2, 3, AVF

T Waves Deeply and symmetrically inverted in leads 2, 3, AVF, high in leads V3 and V4

Interpretation: Tracing consistent with a recent posterior myocardial infarction

CARDIAC DILATATION AND HYPERTROPHY. CONGESTIVE FAILURE. INADEQUATE CARDIAC OUTPUT

CARDIAC DILATATION AND HYPERTROPHY

Dilatation is stretching and weakening of the heart muscle when subjected to infection or unusual strain.

Hypertrophy is thickening of the heart wall caused by increase in the size of the individual muscle fibers

Whenever there is need for increased cardiac output, as during exertion, some degree of dilatation probably occurs, but this is of a physiologic, temporary nature With cessation of added work, the heart returns to normal size without any damage

If extra work is imposed on a heart whose action is handicapped by disease of the muscle, valvulitis, or increased external demand, as in hypertension, pathologic dilatation occurs In acute illness, dilatation may occur suddenly and be severe enough to cause death Frequently the patient recovers and the heart returns to normal size But permanent damage or continuation of abnormal strain will result in progressive changes In such cases or when the initial strain is less acute the first change is some degree of dilatation. As a compensatory response the muscle fibers then become thicker with resultant hypertrophy of the wall and increase of cardiac strength But the larger muscle fibers require more oxygen, and since in hypertrophy there appears to be no increase in blood vessels to supply this demand, the myocardium becomes fatigued and responds by dilating further A vicious circle of dilatation and hypertrophy is thus established, and eventually the point is reached at which further hypertrophy is impossible The heart is then no longer able to maintain adequate circulation and the signs and symptoms of beginning failure appear. A program of rest and other therapeutic measures will relieve myocardial strain and restore muscle tone, dilatation will disappear to some extent, and cardiac efficiency will improve But with resumption of activity, reserve will again diminish, and the amount of exertion necessary to provoke dilatation will decrease. In time, restoration of tone becomes impossible, adequate circula-

tion cannot be maintained even with the patient at rest, and permanent failure is at hand.

In the acute case, cardiac enlargement is the result of dilatation alone. In the chronic case, it stems from a combination of both dilatation and hypertrophy but here it is clinically impossible to determine, in the presence of acute failure, the relative degree to which each exists If the signs of congestive failure can be eliminated by treatment, the teleroentgenogram will probably reveal the degree to which hypertrophy exists uncomplicated by the more transient acute dilatation However, some degree of uncomplicated hypertrophy may exist without being clinically or roentgenologically appreciable

Demonstrable cardiac enlargement is evidence of myocardial injury and, as a general rule, the larger the heart the greater the damage.

Acute Dilatation

Causes

1. Acute myocarditis—infectious, as in rheumatic disease, diphtheria or other infectious disease, or toxic, as in emetine or carbon tetrachloride poisoning.
2. Acute myocardial infarction
3. Prolonged rapid rhythm such as paroxysmal tachycardia, auricular flutter, or fibrillation, especially when it is associated with chronic myocardial disease.
4. Acute high-grade anemia
5. Sudden left ventricular failure from any cause
6. Sudden right ventricular failure, as from pulmonary embolism or an attack of intractable asthma
7. Trauma, as in severe *steering-wheel* injury

Symptoms

In addition to the symptoms produced by the underlying cause, one will find dyspnea or hepatic pain, or both, depending on the side affected

Signs

Altered Apex Impulse. This will be diffuse and extend to the left.

Enlargement. Lateral extension of the left and perhaps the right border will be demonstrable by percussion and x-ray

Murmurs. One or more murmurs not previously present may appear; a blowing systolic at the apex is most likely

Signs of Congestion. Venous engorgement, edema—peripheral or pulmonary—and cyanosis are the rule

Increased Heart Rate. A change of rhythm may also occur

Fall of Blood Pressure. This is usually accompanied by diminished pulse pressure

Change of Heart Sounds. One may find feeble sounds with or without pulsus alternans, gallop, or tic-tac rhythm

X-Ray Findings

Generalized enlargement without characteristic *configuration* is the rule; the shape may reflect the underlying disease. From pericardial *effusion dilatation* is distinguished by the *greater prominence* of pulmonary vessels and, fluoroscopically, the more readily seen cardiac *pulsation*.

Electrocardiographic Findings

In left ventricular strain depressed ST segments and downward T waves appear in lead 1, lead AVL and lateral unipolar precordial leads; in right ventricular strain, the same changes in lead 2, lead 3, and medial unipolar precordial leads. Sometimes ST-T wave changes of non-specific pattern will be found

Whether there is a fatal termination, complete recovery, or a chronically damaged heart depends on the nature and severity of the underlying cause.

Chronic Dilatation and Hypertrophy

There are all degrees of dilatation and hypertrophy, ranging from the slight amount which cannot be determined on clinical examination alone to the huge hearts weighing 700–1000 gm The largest hearts with *respect to hypertrophy and weight* are usually found in free rheumatic or syphilitic aortic regurgitation, rheumatic aortic stenosis, and hypertension of high degree, especially when chronic congestive failure has been present for a long time The largest hearts with respect to volume are found in long-standing mitral disease with predominating stenosis and auricular fibrillation; the left auricle, and perhaps the right ventricle and auricle are enormously dilated, whereas the left ventricle is usually not greatly enlarged Although as a rule the degree of enlargement *can be utilized* as a measure of cardiac damage, *it must be emphasized that serious disease such as angina pectoris or constrictive pericarditis can exist without any appreciable enlargement.*

Dilatation and hypertrophy are often greater on one side than on the other, depending on which is subjected to the greater strain. For example, *increased* tension in the pulmonary circulation brought about by mitral stenosis or some type of chronic pulmonary disease imposes more strain on the right ventricle so that dilatation and hypertrophy will predominate on this side. Aortic val*vulitis or increased peripheral resistance*, as in *hypertension*, by first imposing strain on the left ventricle will cause dilatation and hypertrophy on the left. When the burden becomes too great the left side fails and resultant backing-up of blood in the pulmonary circulation causes dilatation and hypertrophy of the right side If the strain is equally distributed, as in thyrotoxicosis or combined valvular disease such as predominant aortic regurgitation and predominant mitral stenosis, dilatation and hypertrophy will occur on both sides

Causes

1. Vascular hypertension
2. Rheumatic disease. The enlargement may result from rheumatic myo-

carditis, structural valvular disease, or both. Fibrous pericarditis with extensive adhesions may be a contributing factor.

3. Syphilitic aortitis with aortic regurgitation.
4. Coronary disease (sometimes)

Less common causes are thyrotoxicosis, myxedema, chronic bronchopulmonary disease, such as emphysema, asthma or chronic infection, anemia, deficiency states, and congenital defects. In some cases, the cause of cardiac enlargement cannot be determined, even at *post-mortem*.

Symptoms

In the absence of failure, hypertrophy and dilatation usually cause no symptoms. Hoarseness or dry, harassing cough may result from pressure of a dilated left auricle on the recurrent laryngeal nerve or a major bronchus.

Signs

A slightly enlarged heart may give no appreciable physical signs. X-ray may, in borderline cases, prove useful, but the wide variation in the size of normal hearts and thoracic cavities makes it difficult to detect the difference between a normally large heart and one that is only slightly hypertrophied. Assuming that the normal heart weighs 250–300 gm., the presence of enlargement up to 400–450 gm. is difficult to demonstrate by clinical findings. Enlargement sufficient to increase the weight above this figure can usually be determined by the following physical signs.

Modification of Apex Impulse. LOCATION. Barring displacement of the heart from some extracardiac cause, enlargement will be indicated if the apex impulse is found below the fifth interspace or laterally beyond midclavicular line in the fifth interspace. In extremely large hearts the impulse may be in the sixth interspace, rarely, in the seventh.

INTENSITY. In an enlarged heart, except in extreme failure, the apex impulse is more forceful than normal. As a general rule, an increased impulse in the region of the midclavicular line or to the left of it indicates hypertrophy of the right ventricle; a heaving impulse beyond the midclavicular line but below the fifth interspace means a hypertrophied left ventricle.

PULSATION. Visible pulsation is usually seen over a wider area of the chest wall. The wall itself may move with each systole.

RETRACTION. Sometimes there is systolic retraction in the interspaces instead of systolic impulses.

Alteration of Cardiac Borders. If the left border is percussed beyond the midclavicular line in the fifth interspace or more than 4 cm. from the midsternal line in the third interspace or if well-defined dulness is found just lateral to the right edge of the sternum in the third or fourth interspace, enlargement is undoubtedly present. Cardiac displacement or pericardial effusion must be excluded as a cause before enlargement can be assumed. X-ray study is often more useful than percussion.

Change of Heart Sounds. Loud and booming sounds are often heard with cardiac hypertrophy but do not necessarily indicate hypertrophy, since they

also occur with vigorous heart action from exercise or other cause. In dilatation and failure, the sounds may become less intense or change in quality (tic-tac sounds, gallop rhythm), but such variations are not actual evidence of enlargement.

X-Ray Findings

Left-sided enlargement is recognized by rounding of the apex with lateral extension of the left border. Right-sided enlargement, best seen on the lateral view, appears as an extension of the heart shadow anteriorly toward the sternum. Bilateral enlargement shows a combination of these changes.

Electrocardiographic Findings

Predominant enlargement (hypertrophy or strain) of the left ventricle is characteristically indicated by.

1. Evidence of left axis deviation by limb leads
2. Increased amplitude and perhaps slight widening (0 09-0.11 sec.) of QRS complexes.
3. Inversion of T waves and depression with slight upward curving of ST segments in leads 1, 2, AVL, V5, and V6.

When the right ventricle is predominantly affected one finds.

1. Right axis deviation by limb leads

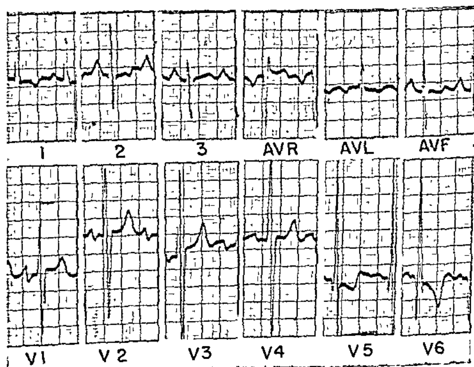


Fig 14 1. Left ventricular hypertrophy due to free aortic regurgitation Left axis deviation Increased amplitude and slight widening of QRS complexes Inversion of T waves and depression of ST segments in leads 1, 2, AVL, V5 and V6.

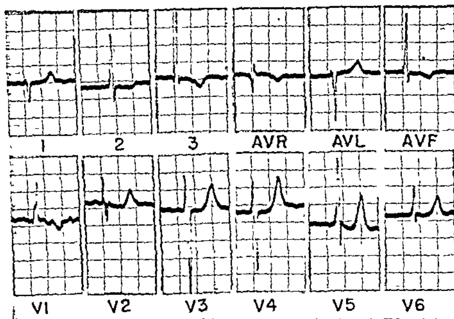


FIG 14 2 Right ventricular hypertrophy due to predominant mitral stenosis Right axis deviation Large R wave in V1 Persistent S wave in left chest leads T wave diphasic in lead 2, inverted in leads 3, AVF and V1 (P waves absent because of associated auricular fibrillation)

2 Increased amplitude of R wave in lead V1 and of S wave in lead V6. QRS voltage as a whole is not usually increased

3. Inversion of T waves and depression of ST segments with slight upward curving in leads 2, 3, V1 and V2.

If both sides are involved a combination of the above changes is the rule.

CONGESTIVE FAILURE

When, because of the strain imposed, the heart muscle is no longer capable of carrying the load, the stage of congestive failure is reached The mechanism underlying the development of this phenomenon is not clearly understood in spite of the efforts of many investigators From two opposing schools of thought have emanated the *forward failure* and the *backward-failure* theories. The former holds that because of myocardial weakness cardiac output becomes incapable of meeting the metabolic demands of the body. Blood flow through the kidneys is consequently reduced, the resultant diminution of glomerular filtration rate causes retention of electrolytes and water in the body The backward-failure proponents contend that incomplete emptying of cardiac chambers due to myocardial weakness results in dilatation of the heart and eventual increase in intracardiac pressure Elevated intra-auricular pressure leads to lowered pressure differential between the venous system and the auricle and hence to impaired auricular filling with consequent stasis in the venous systems Pressure in the capillaries then becomes elevated and loss of fluid and electrolytes into the tissues occurs. In either case certain extra-cardiac factors

such as capillary anoxia, lymphatic obstruction, altered plasma proteins, and *increased blood volume* may also be operative. But irrespective of which theory is correct or whether failure results from a combination of both mechanisms, it is well established that retention of electrolytes and water is the most important factor in congestive failure.

Because left ventricular strain is much more common than right or biventricular strain, the earliest indication of beginning heart failure is almost always inability of the patient to perform without breathlessness activities which *he could formerly accomplish with ease* *Unless due to some non-cardiac cause*, such as poor general muscular condition, obesity, bronchopulmonary disease, or anemia, such breathlessness indicates loss of cardiac reserve. In a case of chronic heart disease it marks the beginning of a period in which this reserve gradually diminishes With time, less and less effort is required to produce shortness of breath. Ultimately the stage is reached at which, even with the patient at rest, the heart is unable to maintain efficient circulation, dyspnea is present *without exertion*, obvious *venous stasis has appeared*, and the patient is in congestive failure.

The side of the heart which bears the greater strain is the first to fail, but it may be impossible to distinguish between right and left-sided failure, especially if at the time he is first examined, the patient is already in a state of advanced failure. The distinction can be made if we know the underlying cause of the heart's weakness—that is, whether the primary strain has been imposed on the right or left ventricle—or if the indications of stasis are limited to the pulmonary circulation or are evidenced in the systemic circulation

To make the picture of right and left ventricular failure and their interrelation as clear as possible, we shall discuss each separately

Left Ventricular Failure

Causes

The most common causes of strain and eventual failure of the left ventricle are.

1. Systemic arterial hypertension of long standing.
2. Disease of the aortic valve—rheumatic, syphilitic or calcareous
3. Insufficiency of the mitral valve.
4. Coronary disease.

Such systemic disturbances as severe anemia, deficiency states, and thyrotoxicosis may effect failure of one or both ventricles

Symptoms

In the early stage, dyspnea on exertion is the important symptom Later it *becomes gradually* more severe, the patient eventually being short of breath even at rest. A type of breathlessness peculiar to left ventricular failure is that known as *cardiac asthma* in which the dyspnea is paroxysmal, not necessarily precipitated by exertion, and often occurs at night, waking the patient out of

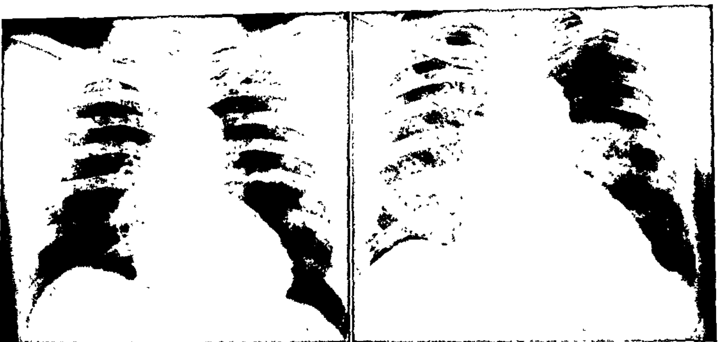


Fig 14 3 Congestive failure due to systemic hypertension

A (*left*) Early Left ventricle enlarged Pulmonary vascular shadows prominent because of engorgement.
B (*right*) Same patient several weeks later with failure more pronounced Further enlargement of heart Prominent hilar shadows Greater prominence of pulmonary vessels Diffuse density of right lung due to pulmonary edema

sleep. Cardiac asthma may appear in advance of any characteristic physical signs and be the first definite indication of impending left-sided failure. Harassing cough resulting from passive congestion of the lungs is also common.

Early Signs

Cardiac Enlargement. In chronic cases this exists before failure sets in; only in acute dilatation can it be looked upon as an actual sign of failure.

Increase of Pulmonic Second Sound.

Decreased Vital Capacity.

Rales in Lungs. Appearing only during an attack of cardiac asthma, these may be *dry, moist, or both*

Later Signs

Rales in Lungs. Moist rales *appear first at the bases*, later throughout the lungs In a long-standing or severe case, edema of one or both lungs may be further indicated by dulness, and diminished fremitus and breath sounds, most likely at the bases.

Pulsus Alaternans.

Protodiastolic Gallop Rhythm. This is best heard in the vicinity of the apex Feebleness of heart sounds, increased heart rate, change of rhythm, or fall in blood pressure may or may not occur

X-Ray Findings

Initially one sees engorgement of the pulmonary vessels. Later hilar and pulmonary vascular shadows become even larger and pulmonary edema appears The latter is indicated by hazy density of the central lung fields, usually *bilateral, but occasionally unilateral or even limited to one lobe* The heart is enlarged, its configuration depending on the underlying disease Additional features of congestive failure which are dependent on right-sided failure are *described below*

Failure of the left ventricle adds strain to the right ventricle, so that eventually the former will be followed by right ventricular failure and the two will coexist

Right Ventricular Failure

Causes

Strain and eventual failure of the right venrticle are most likely due to.

1 Left ventricular failure from any cause
2 Mitral stenosis
3. Types of bronchopulmonary disease causing pulmonary hypertension.
4. Tricuspid insufficiency. This is usually combined with valvular defects on the left side of the heart.
5 Certain congenital malformations, such as interventricular septal defect and pulmonary stenosis.

In children with generalized acute carditis, rheumatic or diphtheritic, failure, when it occurs, usually *predominates on the right side*, probably because the right ventricle is normally the weaker of the two

Symptoms

Pain or discomfort in the right upper quadrant due to passive congestion of the liver is the initial complaint. The symptom may disappear as the disturbance becomes chronic, but the liver will remain enlarged. Dyspnea, although present, is due to the factor causing the right-sided failure, such as pulmonary engorgement in mitral stenosis, or intrinsic bronchopulmonary disease in cor pulmonale.

Early Signs

The early signs of right-sided failure are dependent on stasis in the systemic or hepatic circulation.

Stasis in Systemic Circulation. This is indicated by.

Venous Engorgement. The cervical veins are distended and visible pulsations are evident when the patient is upright (*see* Chap. 8)

Edema In the systemic circulation this usually occurs first in the dependent regions, notably the ankles. Later it appears in the legs, thighs and genitalia; in severe cases it extends upward to include the whole body below the level of the heart and may involve even the neck and arms. Free fluid may collect in one or both pleural cavities (*hydrothorax*), the right usually being affected first. Occasionally hydrothorax appears before peripheral edema Gain in weight may be the first indication of edema.

Cyanosis

Stasis in Hepatic Circulation. This is indicated by:

Enlargement of Liver Except in the long-standing case the liver is also tender.

Enlargement of Spleen This is usually not striking and often not clinically appreciable.

Ascites This occurs only in severe cases

Later Signs

Referable to the heart itself, these may be detectable when failure is well established

Enlargement. As in the case of the left ventricle, enlargement of the right ventricle *per se* is an indication only of previous strain, not of actual failure. It is apparent on physical examination only if it is of considerable degree, that is, enough to displace the apex impulse to the left or, rarely, down and to the left Slight to moderate right ventricular enlargement increases the prominence of the heart anteriorly, but this is difficult or impossible to detect on physical examination Except for the acute dilatation which may occur in acute cor pulmonale, the right ventricle fails only when it is much enlarged Right auricular dilatation is indicated by dulness to right of the sternum

Decrease of Pulmonic Second Sound

Increased heart rate, feeble heart sounds, irregularity, gallop, or tic-tac rhythm may be observed. The last-named will be heard near the lower sternum.

X-Ray Findings

The indications of pulmonary engorgement described above will be present except when the right-sided failure is created by a disturbance which does not cause increased pressure in the pulmonary circuit. Findings attributable to right-sided failure are enlargement of the superior vena cava and azygos vein, enlargement of the liver and, rarely, of the spleen, uni- or bilateral pleural effusion, and sometimes ground-glass appearance on abdominal films indicative of ascites The right side of the heart will be enlarged and, when the failure is secondary to the left-sided strain, the left side also

Two illustrations are given here to clarify the sequence of events which lead up to congestive failure based on right or left ventricular strain

Rheumatic Heart Disease with Mitral Stenosis Free flow of blood from left auricle to ventricle is hampered by narrowing of the mitral orifice. Resultant increased pulmonary vascular tension creates a need for increased work on the part of the right ventricle. This results in dilatation, hypertrophy, and eventual failure of the right ventricle and dilatation of the right auricle The subsequent phase is generalized venous stasis, manifested at first usually by engorgement of the jugular veins, enlargement of the liver, and dependent edema Sometimes hydrothorax appears first When failure becomes sufficiently severe, there will also be generalized edema, hydrothorax, hydroperitoneum, and cyanosis

Because there is no increased load on the left ventricle this chamber remains relatively normal But if appreciable mitral insufficiency is present in addition to the stenosis, there *is* strain on the left ventricle as well as on the right, so that left ventricular dilatation and hypertrophy will occur along with the changes on the right side When failure sets in, both sides of the heart will be affected.

Hypertensive Heart Disease Here the strain is on the left ventricle and this chamber will dilate and hypertrophy as the disease advances When the strain becomes overwhelming, left ventricular failure occurs, manifested by the signs already described. With the left ventricle in a state of failure, the load on the right side is increased, and ultimately we have a completed picture of congestive failure resulting from a disorder which imposed strain primarily on the left ventricle.

INADEQUATE CARDIAC OUTPUT DUE TO CARDIAC DISORDER

The cardiac output or minute volume is the amount of blood ejected into the aorta during each contraction of the ventricles multiplied by the number of beats per minute. In the form of circulatory failure just discussed, the

clinical manifestations are due to engorgement of the venous side of the circulation (pulmonary, systemic, or both), resulting from inability of the heart to function effectively. The volume of circulating blood is *normal* or even *increased* In contrast, a number of disturbances, some unimportant and some serious, are encountered in which circulation is inefficient because of diminished circulating blood volume. In these, cardiac output is decreased because of, (1) some disturbance in the heart itself, or (2) changes in the peripheral vascular system which decrease venous return.

Disturbance of Rhythm

The heart rate may be suddenly slowed in such disorders as irritable carotid sinus syndrome, depression of cardiac pacemakers as by quinidine or of the conduction system as by digitalis, and the *Adams-Stokes* syndrome in severe heart block. Temporary weakness or faintness may be the only symptom, or the patient may fall in faint, either without warning or after a moment of weakness, giddiness, pallor, and sweating Heart rate is slow; blood pressure declines. Circulation shortly returns to normal and the episode is over. If the attack is prolonged, convulsions may ensue. In cases due to serious causes, such as the *Adams-Stokes* syndrome, or where syncope due to a lesser cause is superimposed on underlying serious disease, death may occur.

The various forms of tachycardia—auricular flutter or fibrillation, auricular or ventricular paroxysmal tachycardia—when severe, may be accompanied by weakness, faintness, pallor, coldness, sweating, or nausea Unconsciousness is rare, although mental acuity may be diminished The pulse is thready; blood pressure and pulse pressure are diminished When the disturbed rhythm is of long duration, or when underlying heart disease is present, signs of congestion—dyspnea, orthopnea, rales in the lungs, and distention of peripheral veins—often develop, in contrast to cases of rapid heart action due to *peripheral* circulatory failure, in which these features are absent.

Valvular Disease

In aortic valvular disease, especially predominating stenosis, sudden increased demand on the circulation, as by exertion or paroxysmal tachycardia, may result in temporary faintness or syncope. Similar episodes in severe mitral stenosis tend to be masked by the symptoms due to the marked pulmonary vascular congestion A thrombus in the left auricle in mitral stenosis may temporarily interfere with flow of blood through the mitral orifice and induce faintness or syncope.

Congenital Cardiovascular Defects

Weakness, faintness, giddiness, syncope, convulsions, or coma, may occur following undue strain on cardiac reserve in the more severe forms of congenital cardiovascular disease, especially those in which cyanosis and dyspnea are present

Cardiac Tamponade

In acute cardiac tamponade due to rapidly developing or large pericardial effusion or hemopericardium, inability of the heart chambers to expand reduces intake and consequent output. The clinical picture resembles that of peripheral circulatory failure described below, with the notable exception that the peripheral veins are filled because of hampered cardiac inflow. In constrictive pericarditis, gradually increasing mechanical restriction of inflow and hampered ventricular activity cause diminution of outflow with resultant dyspnea and easy fatigability Peripheral veins are distended.

INADEQUATE CARDIAC OUTPUT DUE TO DEFICIENT CIRCULATING BLOOD VOLUME

In contrast to the above disorders, in which the fundamental disturbance is in the heart or pericardium, here the basic trouble is in the peripheral circulation. Peripheral vasodilatation results in stagnation of blood flow, or blood volume is actually reduced by loss of fluid, plasma or whole blood. Hence, the heart does not receive enough blood to maintain adequate output. This may occur under a wide variety of circumstances; the more severe forms constitute the clinical syndrome known as *peripheral circulatory failure*, or *shock* As a result of studies by many investigators during the past two to three decades, it has become accepted that in general, depending on the fundamental disturbance, there are three different underlying mechanisms outside the heart itself by which peripheral failure may be brought about. These are classified as follows:

1. Neurogenic failure
2. Vasogenic failure.
3. Hematogenic failure.

The forces operating in peripheral circulatory failure are closely interrelated so that in many cases more than one of these mechanisms may be in effect at the same time. Consequently in the forthcoming discussion certain disturbances are listed as responsible for more than one type of failure. It is also possible for circulatory embarrassment to be dependent on insult both to the heart and to the peripheral circulatory apparatus, as for example in acute myocardial infarction or massive pulmonary embolism Here there may be not only myocardial damage or strain but also peripheral changes secondary to reflex disturbance of the vasomotor system In some situations the mechanism underlying the circulatory changes is not clearly understood

Neurogenic Type

Dilatation of certain vascular beds brought about by diminished vasoconstrictor tone is the underlying mechanism Impairment of cardiac efficiency may be a contributing factor. Total blood volume is normal but effective circulating blood volume is decreased.

Causes

The most common are:

1 Emotional stress, such as excitement, fear, or pain.

2 Reflex disturbance of the nervous system, as by a blow on the abdomen, carotid sinus stimulation, or serious insult to any vital structure, as in acute myocardial infarction, large pulmonary embolism, acute pancreatitis, intestinal obstruction with strangulation, perforation of a viscus, or manipulation of organs during an abdominal operation.

3 Direct disturbance of the nervous system, as by spinal anesthesia, trauma, cerebral vascular accident, skull fracture with cerebral injury, drug or chemical poisoning, or, less often, operation on the mid-brain.

The simplest and perhaps most common form is the ordinary fainting attack (*vasovagal syncope*). Occurring usually under sudden emotional stress in a person with an unstable vasomotor system, abrupt loss of vasoconstrictor tone causes pooling of blood in certain areas, fall of arterial blood pressure, and diminished filling of veins, with consequent diminished return flow to the heart. Typically the episode occurs only when the person is upright.

Signs

Unconsciousness. This may appear without warning or following a brief period of weakness, giddiness and faintness.

Pallor. This results from surface vasconstriction

Sweating. In contrast to the more serious late vasogenic or hematogenic form, the skin usually remains warm.

Nausea and Vomiting. These may or may not be present

Slow, Shallow Respiration. Occasionally it is deep and sighing.

Diminished Heart Rate. At the start the rate may be rapid, but shortly becomes slower because of cerebral ischemia or vagus nerve stimulation

Weak Radial Pulse. It is often imperceptible. Pulsations of larger arteries such as the carotids and femorals appear normal

Blood Pressure Changes. Systolic pressure falls rapidly; diastolic may be normal or slightly elevated momentarily, but then falls

If the patient is kept horizontal for a few minutes, normal circulation is re-established and the attack is over. However, when this state is superimposed on serious structural disease such as cerebral or coronary arteriosclerosis or renal insufficiency, death may occur immediately or later.

When due to *reflex* or *direct disturbance* of the nervous system, the early clinical picture is essentially the same. The outstanding features are low blood pressure, weak pulse—either rapid or slow—pallor, sweating, and perhaps nausea and vomiting The skin is warm The patient may be alert and anxious or become unconscious With proper therapy recovery usually takes place; sudden death from vagal inhibition occasionally occurs If proper treatment is not instituted or the cause of the disturbance cannot be corrected, as in cerebral

vascular accident, prolonged vasodilatation and blood stasis will persist and eventually may result in loss of fluid or plasma through damaged capillary walls. Effective blood volume is thus further reduced and the shock state worsens. Moreover, if the injury itself is such as to cause loss of fluid, plasma, or blood, one may have hematogenic shock in addition to the neurogenic failure.

Vasogenic Type

Vascular dilatation is thought to be due, not to nervous system influences, but to *direct injury to the vessels* by some noxious agent

Causes

Among the disturbances regarded as wholly or partially responsible for this form of peripheral failure are:

1. Severe *systemic infection.*
2. Anaphylactic shock.
3. Prolonged deep anesthesia
4. Drug or chemical poisoning

Signs

If the onset is abrupt, as in fulminating meningococcic infection, the picture may at the start resemble neurogenic shock. Otherwise the *initial stage* is characterized by

Restlessness and Anxiety.
Warm, Flushed Skin.
Active Heart with Bounding Pulse.
Low Blood Pressure.

If the process continues, the picture is one of gradual decline and becomes comparable to that described under hematogenic failure with progressive diminution in strength of pulse and blood pressure to the point of imperceptibility, cold, clammy skin, cyanosis which is likely to be blotchy, and other signs indicated below.

Hematogenic Type

Blood volume is diminished by acutal loss of fluid, either blood, plasma, or water and electrolytes. In the past, this type of failure has been referred to as secondary or traumatic shock.

Causes

The common causes are:

1. *Hemorrhage, apparent or concealed.*
2. Capillary injury due to severe burn or some toxic substance, such as one of the snake venoms.
3. Peritonitis.

4. Severe crushing injury.
5. Some metabolic disturbances, such as diabetic acidosis and acute adrenal insufficiency.
6 Severe vomiting, diarrhea or sweating.

Signs

The clinical manifestations of severe peripheral circulatory failure of this type may be summarized as follows:

Weakness, Prostration and Restlessness.

Anxiety. Later apathy, diminished mental acuity, and finally unconsciousness appear.

Anxious, Drawn Expression. The face is often ashen gray and the eyes are sunken

Thirst. This is the single, most striking clinical symptom. Vomiting of ingested fluid is common.

Cold, Ashen-gray Skin. The dependent areas are often mottled The low skin temperature is a result of compensatory vasoconstriction and is important in differentiating between hematogenic failure and early neurogenic or vasogenic failure in which warmth is the rule The skin is often but not necessarily moist. Sluggish capillary circulation can be confirmed by observing failure of nail beds promptly to regain their color following blanching by external pressure

Shallow Respiration. There may be occasional sighing but dyspnea or labored breathing does not occur.

Subnormal Temperature.

Rapid, Thready Pulse.

Rapid, Tic-tac Heart Sounds.

Collapsed Peripheral Veins. They are hard to locate and show poor filling, even after application of a tourniquet.

Blood Pressure Changes. In the early stages, because of compensatory vasoconstriction, systolic blood pressure is normal or only slightly lowered, and the diastolic normal or perhaps elevated Later, both systolic and diastolic pressures fall, the former more rapidly than the latter, thus reducing pulse pressure. In the final stages, readings are unobtainable.

Other important features are changes in the hematocrit, serum protein and specific gravity of whole blood and plasma Their values will be diminished in hemorrhage, because of hemodilution secondary to loss of blood. Following severe burn, in crush syndrome and other disturbances associated with loss of plasma-like fluids and not of whole bood, hematocrit, serum protein, and specific gravity are elevated.

Electrocardiographic Findings

Non-specific ST depression and T wave inversion in several leads may be found in any form of shock.

The manifestations described above represent a composite picture of a late or serious stage No single symptom or sign can be regarded as indicative of the hematogenic type of failure in its incipiency. In any of the potentially serious situations listed above, the likelihood of peripheral failure must be anticipated and treatment initiated without waiting for signs of failure to develop. Successful treatment depends on an accurate appraisal of the nature of the underlying disturbance and, in the individual case, the mechanism by which failure is likely to be initiated If treatment is not started early or is ineffective because of the seriousness of the injury, irreversible changes occur in vital structures presumably as a result of tissue asphyxia, and death eventually results.

Peripheral circulatory failure is differentiated from congestive failure by absence of signs of venous stasis, such as distention of veins, enlargement of liver and peripheral edema, or of dyspnea and rales indicating pulmonary congestion.

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CLASSIFICATION OF CARDIOVASCULAR DISEASE. CONGENITAL CARDIO-VASCULAR DISEASE

CLASSIFICATION

Classification and diagnosis of cardiovascular disease, to be complete, must take into consideration etiology, structural changes, and functional status. A diagnosis of rheumatic heart disease, for example, does not convey all of the essential information. It should be amplified to denote whether the infection is active, whether pericardium, myocardium or endocardium is involved, what valve defects are present, what type of rhythm disturbance exists, whether failure is present or imminent, and the degree to which the patient's activity is or should be restricted For simplicity we have adopted as best for teaching purposes the classification below which is based for the most part on etiology. Naturally, in describing a given case one would include, in addition to etiology, the other factors just mentioned The order in which the disturbances are listed does not indicate their relative frequency or importance.

I Congenital
- A. Acyanotic
- B Cyanose tardive
- C. Cyanotic

II. Infectious
- A. Rheumatic
- B Syphilitic
- C Bacterial
  1. Acute–Staphylococcus, gonococcus, meningococcus, pneumococcus, etc.
  2. Subacute–*Streptococcus viridans*
  3. Diphtheria
  4. Other infections
- D. Rare Types
  1. Trichiniasis

2. Trypanosomiasis
3. Others

III. HYPERTENSIVE
- A. Systemic
- B Pulmonary

IV. CORONARY

V. DISTURBANCES OF RHYTHM

VI MISCELLANEOUS
- A Thyroid disease
- B Anemia
- C. Trauma
- D Nutritional disorders
- E Toxic agents
- F. Neoplasm
- G. Idiopathic cardiac failure
- H Neurocirculatory asthenia
- I. Dissecting aneurysm of aorta

CONGENITAL CARDIOVASCULAR DISEASE

Congenital cardiovascular disease is relatively rare It is found in children more often than in adults, chiefly because the serious anomalies are likely to result in early death The etiology is not always clear But it has recently been well established that if a mother develops rubella during the first trimester of pregnancy, the child is likely to have some congenital defect such as deafmutism, cataracts or other eye disturbance, mental deficiency, or a cardiovascular anomaly

Every infant and young child should have periodic cardiovascular examinations to detect any congenital defect. If a murmur, thrill or other abnormal sign is discovered, the parents should be informed and a record kept If this is done, the same variant found at subsequent examinations will not be erroneously attributed to acquired heart disease. Because of the complexity of many of the lesions, it is frequently impossible by routine clinical examination to determine their exact nature However, two recently developed methods of investigation—angiocardiography, and cardiac catheterization for blood gas analyses and study of circulatory dynamics—have added greatly to diagnostic accuracy. To recognize and differentiate congenital defects is now especially important because of recent advances in corrective and palliative surgery Furthermore, certain anomalies, especially interventricular septal defect, patent ductus arteriosus, and coarctation of the aorta, are particularly susceptible to subacute bacterial endocarditis.

The only defects we shall discuss are those which do not necessarily prevent the individual from reaching late childhood or early adult life and are sufficiently common beyond infancy to present recognizable clinical pictures.

Congenital heart disease, depending on the type and extent of the defects, may exist without any physical manifestations at all, or produce a wide variety

of symptoms ranging from those which offer no interference with normal existence to those incompatible with life.

In such disorders as mild interauricular or interventricular septal defect and uncomplicated patent ductus arteriosus, there may be no symptoms at all More serious defects produce dyspnea and palpitation. The most severe cases show, in addition, headache, dizziness, sometimes convulsions and coma, and symptoms resulting from poor circulation in other parts of the body

Cyanosis is an important feature of the severe cases It is commonly accompanied by *clubbing* of the fingers and toes and by *polycythemia*, which is a compensatory mechanism produced by the relative anoxemia If congestive failure occurs, its characteristic signs complete the picture

Because of the multiplicity and complexity of the defects, classification of congenital heart disease is difficult. We use the Maude Abbott classification[1] which divides the cases into (1) acyanotic, (2) cyanose tardive, and (3) cyanotic.

Acyanotic

Coarctation of Aorta. A rare form usually causing early death in infants is characterized by narrowing of the whole aortic isthmus between the left subclavian artery and the ductus arteriosus. In the more common type, encountered in children and adults, a localized constriction of the aorta exists just beyond, at, or rarely, just proximal to the insertion of the ductus arteriosus. There are all degrees of severity. The aorta is usually dilated above and narrowed below the point of constriction except for a short segment of poststenotic dilatation The proximal enlargement may be enough to cause visible or palpable pulsation above the episternal notch Peripheral blood pressure is increased in the upper part of the body, decreased in the lower Even if, as sometimes occurs, it is not appreciably elevated, *it is always higher in the upper than in the lower extremities*—the reverse of the normal relationship This is virtually a pathognomonic sign

To compensate for the constriction and provide adequate blood supply to the lower parts, extensive collateral circulation develops between the arteries originating above and below the lesion The internal mammary, intercostal, scapular and other arteries increase tremendously in size and may be easily felt; their pulsations are often visible Pulsation of the intercostal arteries not plainly evident may sometimes be detected in the back by pressing against the lower margins of the ribs while the patient is sitting somewhat slumped forward A systolic murmur probably produced at the point of constriction is usually heard not only over the precordium but also over the back and sometimes along the course of the dilated, anastomotic vessels. It may be accompanied by a palpable thrill Cyanosis and clubbing are absent. The heart may be normal or, as a result of the hypertension, may enlarge and eventually fail

X-ray may show diminished prominence of the aortic knob In properly

[1] Abbott M F *Atlas of Congenital Cardiac Disease* New York, American Heart Association, 1936

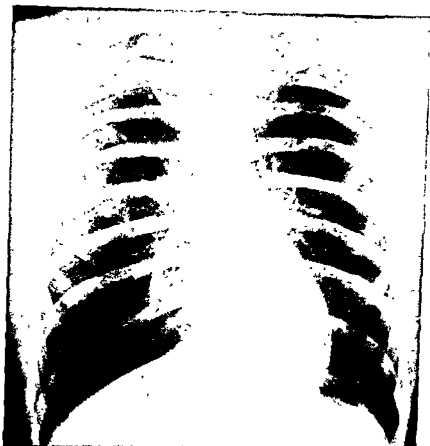


FIG 15 1 Coarctation of aorta Aortic knob less prominent than normal Inferior margins of ribs scalloped

exposed films, indentation of the aortic shadow at the point of coarctation may be visible by contrast with adjacent air-containing lung and barium-filled esophagus. Notching or scalloping of the lower borders of the ribs due to erosion by dilated intercostal arteries is seen when collateral circulation is well established Angiocardiography will confirm diagnosis Electrocardiograms are normal or show left ventricular hypertrophy.

In any case of unexplained hypertension, particularly in a young person, the blood pressure should be taken in the legs as well as the arms to exclude coarctation of the aorta. Because of the potentialities of surgical treatment, early recognition of this defect is of increasing importance

Aortic Stenosis. In rare cases, stenosis of the aortic valve or the infundibulum of the left ventricle 1–2 cm. below the valve may occur congenitally and give rise to signs similar to those of acquired aortic stenosis. If such signs are encountered in infancy or early childhood, congenital origin should always be suspected. Depending on the degree of impairment the characteristic aortic systolic murmur may vary from faint to one which is loud, rasping, and accompanied by a palpable thrill. Aortic second sound is normal, it may be followed by a faint aortic diastolic murmur. Left ventricular enlargement and

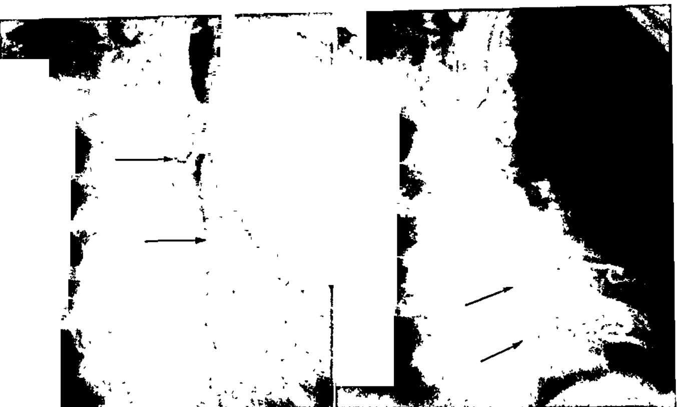


Fig 15 2. Coarctation of aorta

A (*left*). Aortogram following retrograde injection of radiopaque medium into left subclavian artery. Upper arrow indicates point of obstruction, lower arrow, dilated internal mammary artery serving as a collateral channel

B (*right*). Film taken a few seconds after A Arrows indicate dilated and tortuous intercostal arteries

perhaps a small aorta may be noted by x-ray. Electrocardiogram shows left ventricular hypertrophy.

Dextrocardia. The heart may be congenitally misplaced in one of two ways In one type, it is rotated and lies in the right side of the thorax so that the left ventricle is more anterior than normal, but its relationship to the other chambers is unchanged. This variant is apt to coexist with some other congenital anomaly Its importance is commensurate with the seriousness of the associated defect. The other malposition, *mirror type* or *complete situs inversus*, is usually, but not always, associated with transposition of other viscera The heart is in the right thorax with its apex impulse in the right fourth or fifth interspace near mid-clavicular line. The left ventricle is actually on the right side, the right auricle and right ventricle lie to the left and in front of it, toward mid-line Except for the malposition, the heart is normal X-ray and electrocardiogram confirm diagnosis.

In situs inversus, the electrocardiogram shows: Complete inversion of lead 1 and transposition of leads 2 and 3: Lead AVL has the configuration of the normal AVR, and *vice versa*. Precordial leads taken from the usual positions (V1–V6) show the right ventricular (small R, deep S) pattern. However, if precordial leads are taken starting at the *left* of the sternum and progressing laterally across the *right* chest, the normal V1–V6 pattern will be found.

When the heart is discovered on the right side, simple dextrocardia must be excluded before the variant is attributed to marked right-sided enlargement or to displacement caused by an intrathoracic disorder such as massive collapse of the right lung or left pleural effusion.

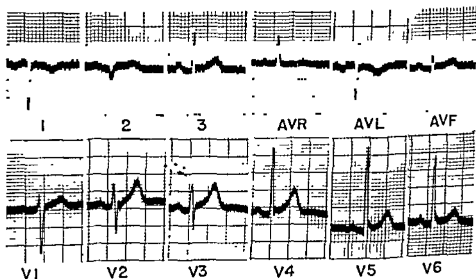


FIG. 15 3 Dextrocardia in a case of situs inversus Limb leads show variants described in text Precordial leads are normal because they were taken with exploring electrode placed to left of sternum for lead V1 and progressively from left to right across the right chest for leads V2–V6

CYANOSE TARDIVE

In congenital cardiovascular anomalies, cyanosis, when present, is usually created by deflection of venous blood into the systemic circulation without passage through the lungs—a right-to-left (veno-arterial) shunt through one of the septa or between the great vessels It is believed that more than 30 per cent of the total blood volume must be diverted to produce an appreciable degree of cyanosis.

In the *cyanose tardive* group, the shunted blood ordinarily passes from arterial to venous side so there is no cyanosis But under excessive strain or in failure, the aberrant flow is reversed; venous blood enters the systemic circulation and cyanosis appears In the *cyanotic* group, the abnormal flow is always from the venous to the arterial side, so that cyanosis is constantly present.

Interauricular Defects. PATENCY OF FORAMEN OVALE. This is probably the most common congenital abnormality and, as a rule, the least important. Normal in fetal life, the foramen ovale permits blood to flow from right to left auricle without passing through the lungs. It closes soon after birth but in some cases a non functioning, valve-like opening persists between the auricles. It is covered by a membrane lying on the left side of the septum, shunt will occur only in severe right heart strain when intra-auricular pressure is greater on the right It is rarely sufficient to cause cyanosis Patent foramen ovale assumes clinical importance in two situations, both of which are rare. An embolus originating in a peripheral vein or the right auricle may pass through the opening and be transported by the systemic circulation to the brain, kidney or some other organ instead of following the usual course into a lung (*paradoxic embolus*) The other complication may occur if, as in severe rheumatic infection, both auricles become greatly enlarged Stretching of the auricular wall will widen the open foramen so that the membrane is no longer adequate A relative defect is thus created, the manifestatoins of which are similar to those encountered in other forms of auricular septal defect

AURICULAR SEPTAL DEFECT. This is relatively large (2–3 cm in diameter). It differs from patent foramen ovale in that there is no covering Rarely, the septum is completely absent The gross defect permits free flow between the auricles. Because of the normally greater pressure in the left auricle, blood passes from *left to right*, thus creating increased load and eventual enlargement of the right side of the heart. Depending on the size of the defect and the pressure differential, the auscultatory findings vary from patient to patient, and from time to time in the same patient They are consequently not as dependable for diagnosis as in most other forms of heart disease There may be no murmurs Or a loud systolic murmur, perhaps attended by a thrill and occasionally followed by a diastolic murmur, may be found to the left of the upper sternum They are thought to be due either to dilatation of the pulmonary artery, or to blood passing through the defect Pulmonic second sound is accentuated Except in the late stages, cyanosis and clubbing are absent, in fact, this is one of the few cardiac disorders in which one finds pronounced right-sided enlargement with-

out cyanosis Terminally, right intra-auricular pressure may become greater than the left; venous blood will then be shunted from *right to left* and cyanosis will appear.

The diagnosis cannot be made except in the presence of characteristic x-ray and electrocardiographic findings. The former shows enlarged pulmonary artery with prominent pulsation, pronounced enlargement of the heart mainly involving the right side, prominent pulsation of hilar shadows (*hilar dance*) and a relatively small aorta Well-marked right ventricular hypertrophy or right bundle branch block is seen on the electrocardiogram.

LUTENBACHER SYNDROME Auricular septal defect is often found combined with some other anomaly. Lutenbacher syndrome—wide auricular septal defect with congenital or acquired mitral stenosis—is the most common The picture is similar to that described above except that one hears the characteristic apical diastolic murmur of mitral stenosis (*see* Chap. 16). X-ray changes are not distinguishable from those of uncomplicated auricular septal defect, but may be more pronounced Electrocardiogram is similar to pure auricular septal defect, except that it may also show broadening and notching of P waves due to left auricular enlargement

Interventricular Defects. ROGER'S DISEASE. Uncomplicated, interventricular septal defect is small and produces no serious symptoms, although the subjects may be relatively short-lived The only clinical sign is a harsh systolic murmur best heard at the third left interspace and often accompanied by a thrill. The murmur is transmitted to left and right and is perhaps heard in the back but rarely in the neck or elsewhere. Shunt is from left to right ventricle but, because the defect is small, disturbance of circulation is minimal Consequently there is no cardiac enlargement, clubbing or cyanosis, x-ray and electrocardiogram are usually normal Diagnosis is made on the basis of the loud murmur without other indications of heart disease, but in childhood it must be made with caution because of the relative frequency, in early life, of loud physiologic murmurs which decrease or disappear later In failure, increased pressure in the right ventricle theoretically should reverse blood flow through the shunt and cause cyanosis, but this rarely occurs Interventricular septal defect is frequently the site of subacute bacterial endocarditis.

EISENMENGER COMPLEX. A dextraposed aorta partially arises from the right ventricle so that a high interventricular septal defect is created An anomaly of the aortic valve resulting in some degree of aortic insufficiency may co-exist. A small amount of blood is pumped directly from the right ventricle into the aorta but may not be enough to cause appreciable cyanosis In most cases, however, cyanosis appears in adolescence or early adult life, varying from day to day but tending to increase with time. A harsh basal systolic murmur accompanied by a thrill is the outstanding sign, it may be widely transmitted One may also hear the characteristic murmur of aortic insufficiency. X-ray shows slight enlargement of both ventricles and perhaps of the pulmonary artery, and prominence and pulsation of hilar shadows Electrocardiogram often shows no characteristic change; in the adult, evidence of right ventricular hypertrophy may

be found. Diagnosis can be definitely established only by cardiac catheterization and angiocardiography.

Interventricular septal defect is often associated with dextraposition of the aorta and pulmonic stenosis (*tetralogy of Fallot*), an anomaly which may be confused with Eisenmenger complex. Here, by x-ray, the right side of the heart is slightly enlarged, the pulmonary artery normal or small, and hilar shadows are not prominent. Electrocardiogram shows right ventricular hypertrophy.

Patency of Ductus Arteriosus. The ductus arteriosus is the structure which, in fetal life, permits blood to flow from pulmonary artery into aorta without passing through the lungs. Normally it closes shortly after birth, but in a few persons it remains patent. After birth, aortic pressure is higher than pulmonary pressure. Consequently, blood flow through a patent ductus is from the aorta back to the pulmonary artery.

The chief finding is a murmur loudest in the second left intercostal space, but sometimes heard all over the precordium and even medial to left midscapula. It is continuous, with systolic accentuation, except in infancy and early childhood, when it may be only systolic. It has a characteristic quality, closely resembling humming machinery and when present is virtually pathognomonic. An accompanying thrill may be palpable. This murmur must be distinguished from the venous hum often heard above the clavicle, especially in children (*see* Chap. 12). Venous hum varies in intensity with changes in the position of the neck and may be obliterated by jugular compression. *An intense murmur does not indicate a severe defect*, on the contrary, the narrower, and consequently the less important the opening, the louder might be the murmur.

The other manifestations of patent ductus depend on the caliber of the anomaly. When the patency is small and unaccompanied by other defects, it is of no mechanical consequence. With a wider channel, enlargement of both ventricles may result from the extra work imposed. Dyspnea and palpitation appear but since the shunt is from the systemic to the pulmonic circuit, cyanosis and clubbing are absent. Diastolic pressure is low, pulse pressure is high. These changes may be sufficient to cause Corrigan pulse and other peripheral vascular signs similar to those encountered in free aortic regurgitation. Terminally, pressure on the right may exceed that of the left, whereupon shunted blood will flow from pulmonary artery to aorta and cyanosis will appear. Radiographic study is helpful except when the shunt is minimal. It shows prominence of the left upper border of the heart shadow due to dilatation of the pulmonary artery resulting from its increased load, but without as much left auricular enlargement as seen in mitral stenosis. The ventricles are enlarged, hilar shadows prominent and often dancing. Electrocardiogram may be normal or show left or right ventricular hypertrophy.

Patent ductus arteriosus is especially liable to invasion by *Streptococcus viridans* with resultant subacute bacterial endarteritis comparable to endocarditis.

Pulmonic Stenosis. This lesion is characterized by a systolic murmur and thrill of maximum intensity in the pulmonic valve area, and hypertrophy of the right side of the heart. Cyanosis may be present in the late stage. It suggests

associated patency of the interauricular septum but may be due solely to insufficient pulmonary blood flow. X-ray may show enlargement of the right auricle and ventricle; the pulmonary artery shadow is often very large due to dilatation beyond the stenosis. Right ventricular hypertrophy of high degree is the important electrocardiographic finding.

Cyanotic

Tetralogy of Fallot. This is probably the most common cause of marked congenital cyanosis seen beyond early life. It is a combination of pulmonary stenosis, ventricular septal defect, right ventricular hypertrophy, and dextraposition of aorta. Cyanosis is produced in the following manner: Some of the blood which has entered the right ventricle, being delayed by the pulmonary stenosis, is pumped directly into the aorta which overrides the ventricular septum and communicates with the right ventricle as well as the left. The added burden on the right side of the heart creates right ventricular hypertrophy, which may be so pronounced as to cause bulging of the left chest wall. Clubbing, retarded growth and polycythemia result from the persistently diminished oxygen content of the blood. Dyspnea on mild exertion is pronounced. A loud systolic murmur accompanied by a thrill is heard over the precordium with maximum intensity along the left sternal border. In contrast to the murmur of aortic stenosis it is not transmitted to the neck. The pulmonic second sound is weaker than the aortic, the reverse of the usual relationship in the young. However, if the aorta is displaced far to the left, the aortic second sound may be best heard to the left of the sternum and be wrongly interpreted as a loud pulmonic second sound. X-ray shows slight enlargement of the right ventricle with diminished prominence of pulmonary artery, and normal or small hilar shadows. Electrocardiogram shows moderate or marked right ventricular hypertrophy.

Transposition of Great Vessels. The aorta arises from the right ventricle, the pulmonary artery from the left. This anomaly is obviously incompatible with life unless some other defect permits some of the venous blood to reach the lungs. Survival is possible if there is a patent foramen ovale, interventricular septal defect, or persistent ductus arteriosus, which will permit some of the venous blood to reach the lungs and some of the oxygenated blood to enter the systemic circuit. Very few cases survive more than a few months or years. Dyspnea, cyanosis, and clubbing appear early and are usually pronounced. The heart is enlarged. Murmurs, other local signs and x-ray findings depend on the nature of the associated defect. The most characteristic findings are narrowing of the great vessel shadows in the anteroposterior projection, widening in the lateral projections and decided prominence of hilar shadows. The heart is enlarged, especially on the right. Electrocardiogram shows right ventricular hypertrophy.

Reference

1. Abbott, M. E.: *Atlas of Congenital Cardiac Disease.* New York, American Heart Association, 1936.

RHEUMATIC HEART DISEASE

RHEUMATIC INFECTION

The changes brought about in the heart by different infectious diseases are so varied that they must be discussed separately. Rheumatic heart disease is the most common Although most cases of this malady are attributable to previous rheumatic infection, a considerable number follow acute tonsillitis, scarlet fever or chorea and some give no history of antecedent infection But since these are indistinguishable clinically and pathologically from cases in which the relationship to rheumatic infection is well-established, they too are regarded as rheumatic heart disease Rheumatic infection, and rheumatic heart disease irrespective of its antecedent history, are now generally thought to be precipitated by preceding infection with beta hemolytic Streptococcus of certain strains In a doubtful case, demonstration of antistreptolysin or other streptococcal antibody in the blood would favor a recent streptococcal infection and indirectly support a diagnosis of rheumatic fever.

Acute Rheumatic Infection

The typical attack of rheumatic fever, almost always preceded by acute tonsillitis or acute upper respiratory infection, produces fever, intense pain in one or more joints, and, when severe, periarticular redness, swelling, and tenderness It is characteristic for the joint symptoms to migrate rather quickly from one joint to another, but this is by no means essential for diagnosis. The acute case may appear to subside within a few weeks; actually infection may smoulder for months or years manifesting itself by prolonged ill-health, failure to gain weight, and possibly, slight, persistent fever. Frequently, especially in young patients, the disease begins insidiously and pursues a less acute course with lassitude, easy fatiguability, loss of appetite and possibly of weight Joint or limb pains may be fleeting or mild or may not appear at all

The extent of cardiac damage cannot be judged by the severity of fever or joint symptoms Because of its very obscurity, the mildly active case often develops serious carditis before being discovered, and treatment is instituted too late Hence, the younger patient with less obvious rheumatic infection may suffer more serious heart damage than the one who, at onset, develops the acute polyarticular form.

It is very important to appreciate the distinction between rheumatic in-

fection as it occurs in youth and as it affects the adult. In youth, one is dealing with active infection; our primary concern is the effect of this infection on the individual as a whole and on the heart muscle in particular. Valve lesions may be present but, at this stage, are of only secondary importance *The severe case of rheumatic heart disease in the young is one of overwhelming rheumatic infection affecting chiefly the myocardium and often the pericardium and endocardium as well,* sometimes pleura and lungs are involved. Death, if it occurs, is the result of this overwhelming infection.

The initial attack of rheumatic infection is less common in adults. Usually onset has occurred earlier and by the time adult life has been reached, the infectious process is less obvious However, exacerbations of an acute nature are common They are usually precipitated by an attack of acute upper respiratory disease, most likely due to the beta hemolytic Streptococcus. When onset does take place in adult life, the illness as a rule is acute and joint involvement is greater but recovery is apt to be more rapid and complete Consequently in the adult, emphasis is on the mechanical handicap caused by structural damage to the heart resulting from infection in earlier years although susceptibility to exacerbations is an added hazard. Valvular deformity with perhaps myocardial damage or extensive adhesive pericarditis has impaired cardiac efficiency The heart eventually cannot overcome the mechanical handicap, and death occurs from congestive failure However, failure and death are not always entirely attributable to structural changes; they may be hastened by recrudescence of active disease or by one or more pulmonary emboli, to which patients in partial failure are especially susceptible Another danger is subacute bacterial endocarditis superimposed on an already damaged valve; this can occur either before or after the stage of mechanical failure has been reached Formerly almost inevitably fatal, this complication is now happily often cured by antibiotic therapy.

Smouldering Rheumatic Infection

Every case of rheumatic infection must be looked upon as a potential case of rheumatic heart disease Only one in every four or five cases escapes demonstrable cardiac damage Hence, evidence of persistent trouble is almost enough to justify the assumption that the heart is involved, even though significant cardiac signs are lacking In the less evident case, presence of continued low-grade infection is indicated by one or more of the following manifestations:

Symptoms

Aches and Pains in Joints or Limbs. Redness, swelling or tenderness may or may not appear.

Fever. A daily rise of rectal temperature to over 100°F. is regarded as fever.

Signs

Recurrent Epistaxis. Trauma or some local lesion of the nose must be excluded.



FIGS 16 1 Erythema marginatum, unusually pronounced, in a case of active rheumatic infection (Courtesy Dr T Duckett Jones)

Rheumatic Nodules. These appear as non-tender, painless, subcutaneous lesions found most commonly along the tendons and over bony prominences on the back of the hands, elbows, knees or spine (*see* Chap 6).

Transitory Skin Eruption. The marginatum form of erythema multiforme is the most common (*see* Chap 3)

Active Chorea.

Failure to Gain Weight.

Anemia.

Persistently Elevated Leukocyte Count. As a rule, it is over 10,000.

Persistently Increased Sedimentation Rate.

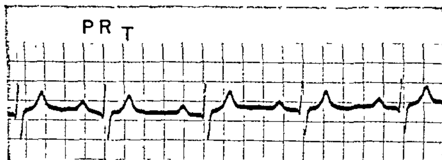


FIG 16 2 Prolongation of auriculoventricular conduction time (P-R interval 0 24 sec) in a case of active rheumatic infection. Lead 2 (Courtesy Dr Benedict Massell, House of The Good Samaritan)

Electrocardiographic Changes

First or second degree heart block is common Alteration of T waves or, rarely, of QRS complexes may be found. Any of these is an indication of carditis (*see below*).

CLINICAL ASPECTS OF RHEUMATIC HEART DISEASE

When rheumatic infection invades the heart, the myocardium, endocardium and pericardium are all probably affected. Exactly to what extent each has been damaged, particularly in the early stages, is often impossible to determine. Unless there is some unmistakable indication of such damage, rheumatic carditis cannot be proved but prolonged active rheumatic infection is strong presumptive evidence

Rheumatic Myocarditis

In rheumatic heart disease, this is one of the important factors affecting cardiac efficiency. In the young it is the chief cause of circulatory embarrassment or failure. In older persons, although the emphasis is on the handicap imposed by chronic valvular and perhaps pericardial changes, the effect of the disease on the muscle cannot be disregarded. Some patients with marked chronic valvulitis or pericarditis will live for years without showing signs of circulatory impairment, while others with comparable valvular or pericardial changes will develop failure at an early age In the first group, the myocardium has been less seriously damaged, the heart is better able to withstand through the years the strain imposed by the other defects.

Rheumatic Endocarditis

The diagnosis of valvulitis cannot be made in the early stage of rheumatic infection. The murmurs and other signs indicative of valvulitis do not appear until scarring and perhaps calcification have advanced to the point of causing stenosis or regurgitation. Murmurs similar to those of valvulitis may appear during the acute stage but these are usually the result of relative stenosis or regurgitation based on dilatation due to myocarditis They often disappear *when the myocarditis improves and muscle tone is restored.*

In the *aortic* valve, thickening of the cusps, adherence of adjoining cusps at their commissures, and retraction and fixation of leaflets prevent proper closing of the valves and result in varying degrees of regurgitation Scarring and calcification may, by fixing the leaflets, obstruct the lumen and produce stenosis. The *mitral* valve is involved twice as often as the aortic. The pathologic process is essentially the same Contraction and rigidity of the chordae tendineae contribute to fixation of leaflets. The pulmonic and tricuspid valves may show similar changes but are less often affected. The relative degrees of stenosis and regurgitation vary widely in different cases and, in the individual case, change with advance in the process. Sometimes there will be almost pure regurgitation, sometimes almost pure stenosis, but where either exists the

very nature of the process is such that the other is almost always present to some extent.

Rheumatic Pericarditis

At first this appears as *fibrinous* (*dry*) pericarditis The exudate may be limited to a few small areas or be extensive An effusion may develop (*serofibrinous* pericarditis) but clinically recognizable pericardial fluid is not common in rheumatic disease Following the acute stage, the pericardium may return to normal More likely small areas of fibrous thickening will form on the pericardial surfaces or light, innocuous adhesions will develop between them The latter are sometimes extensive and may even obliterate the pericardial sac, yet appear to have produced no cardiac embarrassment. Another and more serious possibility is dense thickening of the pericardial membranes with obliteration of the cavity and, sometimes, fixation of the heart to the chest wall, diaphragm, or mediastinal structures Pericarditis is discussed more fully in Chapter 23.

Early Manifestations

As noted above, definite evidence of rheumatic carditis in the early stages is usually provided by the myocardium; dependable signs of endocarditis do not appear until later. If myocarditis cannot be demonstrated, one cannot be sure of carditis until structural valve changes sufficient to cause signs have occurred Pericarditis extensive enough to produce signs is unlikely to occur except in conjunction with clinically evident myocarditis. The earliest sign of myocardial disease is often provided by the electrocardiogram The most common change is prolongation of auriculoventricular conduction time; alterations of QRS complexes or T waves are sometimes significant but the latter may be dependent on pericardial rather than myocardial involvement Since the electrocardiographic variants are likely to be transient they may be overlooked unless serial tracings are taken Some observers utilizing frequent tracings have reported abnormalities in as many as 90 per cent of cases studied.

Signs of Rheumatic Myocarditis

If repeated electrocardiograms fail to show changes, the diagnosis of myocardial involvement depends on one or more of the following signs

Enlargement on Physical or X-ray Examination.

Appearance of Murmurs. Based on relative insufficiency or stenosis of one or more valves, a systolic blow or mid-diastolic rumble in the mitral area is most likely. In more severe cases there may also be a systolic or diastolic murmur at the base. Sometimes a harsh systolic murmur appears in the pulmonic area due to dilatation of the pulmonary artery from increased pressure in the pulmonary circulation. These murmurs disappear if, with improvement, muscle tone is re-established. The mistake of attributing them to structural valvulitis is often made.

Disturbance of Rhythm. Gallop rhythm or persistent simple tachycardia is the most likely. Occasionally auricular fibrillation or flutter may appear.

In its most serious form, rheumatic myocarditis quite suddenly produces, usually in a child or young adult, the signs of severe cardiac embarrassment: dyspnea, distended veins, enlarged liver and perhaps edema. Febrile response becomes greater. Precordial ache is an occasional symptom occurring even in the absence of demonstrable pericarditis. When it is this severe the myocarditis is almost certain to be accompanied by widespread involvement of the other layers of the heart (*pancarditis*) Death from heart failure may occur. The pancarditis is occasionally accompanied by rheumatic pleuritis, rheumatic pneumonia or, rarely, rheumatic peritonitis (*see* Chap 29).

Later Manifestations

The diagnosis of rheumatic carditis in its later stages depends chiefly on the signs produced by the structural valvular defects. Since in rheumatic heart disease pure stenosis or pure regurgitation rarely exists, changes are more correctly described in terms of the valve affected rather than in terms of the defect One should think and speak of rheumatic aortic valve disease or rheumatic mitral valve disease However, it is almost always possible to determine which deviation predominates, so one can qualify the term further and specify, for example, *rheumatic mitral disease with predominating stenosis* or *rheumatic aortic disease with predominating regurgitation* For brevity, common usage permits such terms as *mitral stenosis* and *aortic insufficiency* to indicate the major variant.

The most important defects, in the order of their frequency are:

1. Mitral disease alone
2. Mitral disease accompanied by aortic disease, with predominating regurgitation
3. Aortic disease alone, with predominating regurgitation

Disease of the tricuspid valve frequently accompanies that of the aortic and mitral, by which it is usually overshadowed Serious rheumatic disease of the pulmonic valve is rare

Identification of valvular lesions depends on the physical, radiographic, and electrocardiographic signs The symptoms, barring actual rheumatic disease of the myocardium itself, are dependent on the extent to which the valvular changes hamper cardiac efficiency With lesser degrees of stenosis or regurgitation, the mechanical handicap may be so slight that symptoms and cardiac enlargement are absent or minimal With more pronounced valvular damage, the burden will be greater, hypertrophy and dilatation will occur, and the manifestations of cardiac inefficiency will become progressively more evident Dyspnea and palpitation produced by less and less effort as times goes on are the usual initial symptoms, although abdominal discomfort due to hepatic congestion, or peripheral edema may appear first.

RHEUMATIC VALVULAR DISEASE

Mitral Disease With Predominating Regurgitation

Signs

Murmur. The characteristic murmur of structural mitral regurgitation is a systolic murmur heard over the lower precordium, loudest at the apex. If sufficiently intense it will be audible over a wider area of the chest and in the back. Since the apical systolic murmur has a variety of causes, by itself it can be considered a sure indication of mitral disease only when it is loud, harsh, and not more readily accounted for by some other disturbance Otherwise, mitral regurgitation can be assumed only when the murmur is found in conjunction with the signs of mitral stenosis, with cardiac enlargement not explained by some other factor such as hypertension, or when there is a history

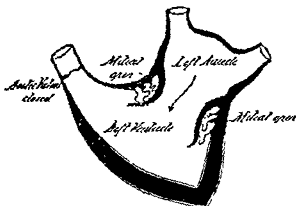


Fig 16 3 Position of valve leaflets in normal heart during diastole Mitral valve open, permitting blood to flow from left auricle into left ventricle Aortic valve closed

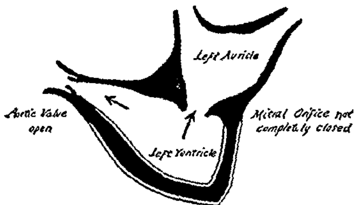


Fig 16 4 Position of valve leaflets during systole in mitral regurgitation Mitral leaflets do not completely close the orifice, thus permitting some blood to flow backward from left ventricle into auricle Blood also flows in normal direction through open aortic valve.



FIG 16 5 Systolic murmur at apex in a case of *early rheumatic mitral disease with regurgitation*



FIG 16 6 Sounds at apex in a more advanced stage of mitral regurgitation. Systolic murmur is louder and masks the first sound In this and succeeding diagrams hollow block indicates a masked sound

FIG 16 7A Typical findings in well-established mitral regurgitation

Horizontal lines represent systolic murmur In this and subsequent diagrams, a murmur is usually best heard in region indicated by a dot but is rarely confined to such a limited area

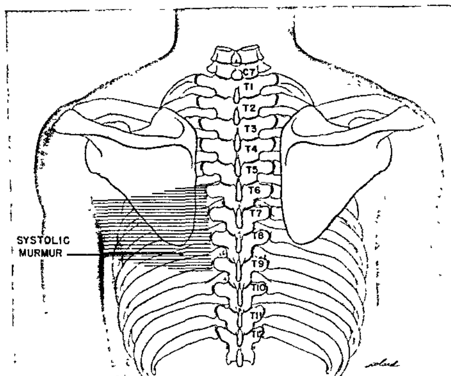


FIG 167B Typical findings in well-established mitral regurgitation When pronounced, the murmur is heard in the back

of prior rheumatic infection. In the last instance the existence of mitral regurgitation is doubtful unless some of these other criteria are present Pure clinical mitral regurgitation is seen in children mostly; it is rare in adults

Cardiac Enlargement. Enlargement of the left auricle occurs first. Sometimes it is demonstrable by percussion, more often only by roentgenogram and flouroscopy Later there will be dilatation and hypertrophy of the left ventricle and, as the disease advances, enlargement of the right ventricle and auricle.

Increased Pulmonary Second Sound. This is dependent on increased pressure in the pulmonary circulation and is most evident when the initial stage of failure is reached.

X-Ray Findings

The left auricle is enlarged and on fluoroscopy, may show prominent expansile movement at the moment of ventricular systole—indication of regurgitation through the mitral valve The left ventricle is enlarged In the late stages, the pulmonary vessels become engorged and the right ventricle enlarged.

Electrocardiographic Findings

P waves may be increased in size and left ventricular hypertrophy may be evident.

Mitral Disease With Predominating Stenosis

Signs

Murmur. Minimal or early mitral stenosis is indicated by an apical mid-diastolic murmur, rumbling or rolling in quality. Usually it is heard more clearly with the patient recumbent or on his left side, and may be detected only by the bell-shaped receiver and sometimes only after moderate acceleration of the heart by exercise. *It may be preceded by a faint third sound.* With an exceptionally rapid rate it can be overlooked until the heart is slowed. The apical systolic blowing murmur of mitral regurgitation may also be present.

In a more well-established case, the murmur is louder, low-pitched, and the

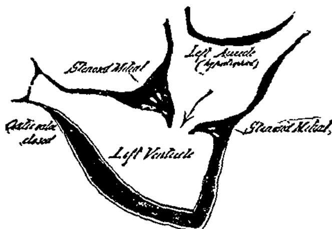


Fig. 16 8 Position of valves during diastole in rheumatic mitral stenosis Thickening and fixation of mitral leaflets prevent free flow of blood from left auricle into ventricle Aortic valve closed

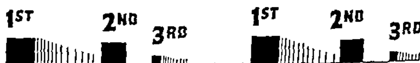


Fig 16 9. Murmurs at apex in rheumatic mitral disease with regurgitation and early stenosis Loud systolic murmur, audible third sound, and faint mid-diastolic murmur Sounds at base normal or pulmonic second sound somewhat increased.



Fig 16 10 Sounds at apex in well established rheumatic mitral disease. Systolic murmur of regurgitation Loud crescendo mid- and late diastolic murmur of stenosis. First sound at apex loud and snapping Pulmonic second sound increased.



FIG 16 11 Sounds at apex in advanced rheumatic mitral disease with predominating stenosis Loud mid- and late diastolic murmur and sharp first sound Palpable diastolic thrill usually present. Systolic murmur of mitral regurgitation may or may not be present Pulmonic second sound loud and usually reduplicated

presystolic phase becomes accentuated, giving a crescendo effect. The first sound at the apex has a sharp, snapping quality. In advanced cases, the murmur is definitely loud and occupies most of diastole. The first sound at the apex is loud and snapping. The systolic apical murmur usually remains, but is apt to be faint.

In the presence of auricular fibrillation the characteristic murmur of mitral stenosis may be much less evident (*see below*).

FIG 16 12 Typical findings in well-established mitral disease with predominating stenosis

Thrill. *The diastolic murmur in the well-advanced case* is accompanied by a palpable thrill over lower precordium. This is a confirmatory sign. Proper timing of the thrill may be impossible with rapid heart rate. It can be confused, especially in a thin person, with the systolic thrust imparted to the chest wall by an overactive heart.

Cardiac Enlargement. The left auricle enlarges first. Initially enlargement is demonstrable only by x-ray; later it can be found by percussion. It is an important confirmatory sign As strain on the right side of the heart increases, enlargement of the right ventricle and auricle develops. In the absence of appreciable mitral regurgitation, aortic disease, or some other factor imposing strain on the left ventricle, this chamber does not enlarge.

Accentuation of Pulmonic Second Sound. In advanced cases it may be followed by a short diastolic blowing murmur due to relative insufficiency of the pulmonic valve (*Graham Steelle murmur*).

X-Ray Findings

Enlargement of the left auricle is predominant. It can be observed fluoroscopically or on films in the lateral or oblique projections. When pronounced, it can be seen through the heart shadow centrally in properly penetrated sagittal projection films. *Calcification in the valve may be detected In tight* stenosis, the left ventricle is rarely appreciably enlarged. Indications of pulmonary vascular engorgement, edema of lungs, and right ventricular enlargement appear eventually.

Electrocardiographic Findings

Wide and macroscopically notched P waves, right axis deviation, and right ventricular hypertrophy are characteristic.



Fig 16 13. Predominant mitral stenosis

A P A film showing fullness of upper *left cardiac border* due to enlargement of left auricle

B Lateral film showing posterior displacement of barium-outlined esophagus by enlarged left auricle.

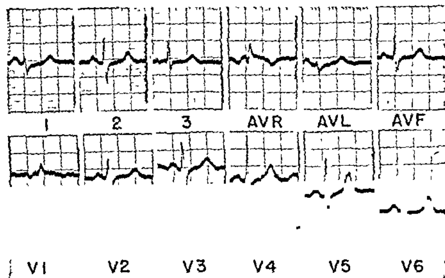


FIG 16 14 Predominant mitral stenosis P waves wide and notched Right axis deviation

Additional Features

In moderate and severe cases one may find

Slight Cyanosis of Lips and Cheeks. This gives the patient a typical facies (*malar flush*) often so distinctive that it can be promptly identified by the experienced observer

Chronic Passive Congestion of Lungs. This is often demonstrable on physical examination but sometimes only by x-ray. In some patients, transitory episodes of acute pulmonary edema occur; they are often accompanied by hemoptysis The latter is much less likely in pulmonary edema based on other forms of heart disease

Signs of Venous Stasis. Distention of neck veins, enlargement of liver, and peripheral edema are indications of right-sided failure secondary to the pulmonary hypertension and engorgement

Auricular Fibrillation. This is common in well-established mitral disease with predominating stenosis, especially when the left auricle is appreciably enlarged Conversely, in rheumatic heart disease, this rhythm is most often associated with mitral stenosis, hence its presence in known rheumatic heart disease strongly suggests mitral disease In uncontrolled auricular fibrillation because of absence of auricular systole, the murmur of mitral stenosis may not show its typical characteristics. Presystolic accentuation of the murmur may be absent, only an early diastolic murmur may be heard or sometimes no murmur will be audible However, if the ventricular rate is controlled by digitalis, a rumble in diastole will usually become evident In a doubtful case, diagnosis must be made on the basis of other indications: rheumatic history, snapping quality of the apical first sound, roentgenologic appearance of the heart, and electrocardiographic evidence of right ventricular hypertrophy

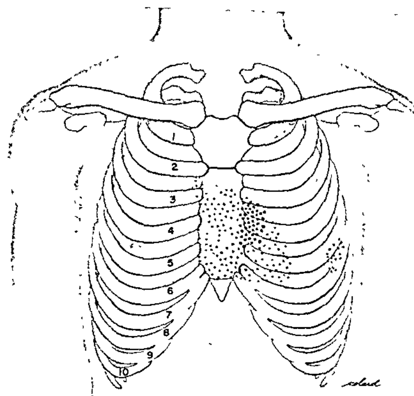


Fig 16 16 Murmur of aortic regurgitation Heavy dots indicate area in which it is usually best heard



Fig 16.17 Rheumatic aortic disease with slight regurgitation A diastolic murmur follows second sound

most important causes of great left ventricular dilatation and hypertrophy. If considerable aortic stenosis exists along with the regurgitation, moderate enlargement is the rule. In either event, enlargement of the other chambers eventually occurs as a result of chronic strain. In the lesser stages of aortic regurgitation, there may not be sufficient impairment of function to produce left ventricular strain and enlargement In these cases the diagnosis is made solely on the character of the murmur.

Peripheral Vascular Changes These depend on *free* aortic regurgitation, are purely confirmatory, and are not diagnostic requirements Rarely, if other

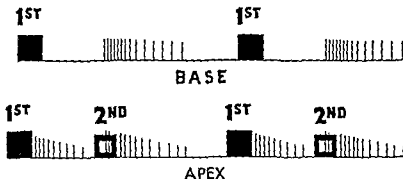


Fig 16 18 Rheumatic aortic disease with free aortic regurgitation At base and along left border of sternum a loud diastolic murmur replaces second sound At apex the same diastolic murmur is heard, it may mask second sound An apical systolic murmur due to relative mitral insufficiency is usually present

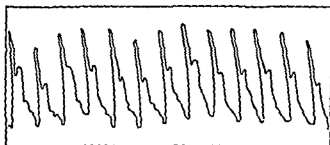


Fig 16 19 Sphygmographic tracing of collapsing pulse in advanced aortic regurgitation

signs are inconclusive, the peripheral changes may be helpful in distinguishing between aortic and pulmonic regurgitation

1. Suprasternal pulsation Throbbing of the aorta or carotid arteries may be seen or felt in or near the suprasternal notch This phenomenon is also common in hypertension and may be encountered in advanced arteriosclerosis and aneurysm

2 Visible pulsation with characteristic jerking of the carotid, subclavian and other larger arteries The carotid artery movement may be sufficient to make the patient's head shake synchronously with cardiac systole

3 Low diastolic with consequent increased pulse pressure. Systolic pressure is often elevated also.

4 Corrigan pulse (*see* Chap 9)

5 Capillary pulse (*see* Chap 8)

6 Pistol shot sound (*see* Chap 12)

7 Duroziez's sign (*see* Chap 12)

X-Ray Findings

The aorta is elongated, tortuous and, on fluoroscopy, shows a collapsed type of pulsation The heart is enlarged downward and toward the left.

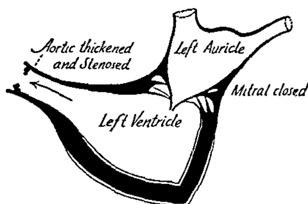


FIG 16 20 Position of valves during systole in aortic stenosis Flow of blood from left ventricle into aorta impeded by narrowing of aortic orifice Mitral valve normally closed but may be relatively *insufficient because of left ventricular dilatation*

Electrocardiographic Findings

Evidence of left ventricular hypertrophy is the rule, except when the leak is minimal

AORTIC DISEASE WITH PREDOMINATING STENOSIS

Signs

Murmur. Characteristically, this is loud, rough, systolic, best heard in the second intercostal space just to the right of or beneath the sternum, and usually transmitted along the vessels into the neck and downward along the left sternal border. It usually masks the first and sometimes the second sound in the aortic area In some cases the murmur is not harsh It is often accompanied by the murmur of aortic regurgitation Occasionally, transmission is downward as well as upward, and if the murmur reaches the apex a mistaken diagnosis of mitral regurgitation may be made In mitral regurgitation, however, the murmur is usually audible in the axilla and posteriorly over the base of the left lung, in aortic stenosis it is not.

Diminished Aortic Second Sound. This sound is usually diminished or absent but if it is normal, the diagnosis of aortic stenosis is not necessarily invalidated

Systolic Thrill. Accompanying the murmur in about one half of the cases, this thrill is felt over the base of the heart, especially over the aortic valve area or in the suprasternal notch

Cardiac Enlargement. Left ventricular enlargement is the rule, but it is usually not as marked as in free aortic regurgitation. The left auricle and later the right chambers enlarge as the disease progresses

Plateau Pulse. This may be recognizable on palpation but frequently can be demonstrable only with *the sphygmograph (see Chap 9).*

Low Pulse Pressure. In well-established cases, there may be a spread of not over 15–20 mm. Hg between the systolic and diastolic levels

MAXIMUM INTENSITY OF SYSTOLIC MURMUR AND THRILL

LEFT VENTRICLE SOMEWHAT ENLARGED

FIG 16 21 Location of systolic murmur and thrill in aortic stenosis (Horizontal lines) Often the murmur is audible over a wider area

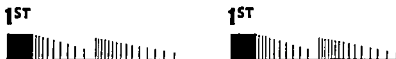


FIG 16 22 Sounds at base in rheumatic aortic disease with regurgitation and beginning stenosis A rough systolic murmur follows the first sound, a diastolic murmur replaces the second They may or may not be heard at apex



FIG 16 23 Sounds at base in rheumatic aortic disease with marked aortic stenosis A loud rough systolic murmur masks the first sound Second sound usually diminished Diastolic murmur of regurgitation may or may not be present Systolic murmur is often transmitted to apex Systolic thrill usually palpable



FIG. 16 24 Sphygmographic tracing of plateau pulse in well-established aortic stenosis

X-Ray Findings

Fluoroscopically the aorta shows pulsation of diminished amplitude. It is not significantly enlarged or tortuous Calcium deposits may be detected in the valve. The left ventricle is enlarged

Electrocardiographic Findings

Left ventricular hypertrophy is evident

Diagnostic Pitfalls

Syphilitic Aortitis with Aortic Regurgitation. (*see* Chap 17).

Calcareous Aortic Disease. Occurring in older men, less often in older women, this is marked by extensive fibrosis and calcification of the aortic valve with pronounced stenosis and usually slight subclinical regurgitation. Other valves

FIG 16 25 Aortic stenosis and regurgitation Electrocardiogram shows pronounced left ventricular hypertrophy

are not involved Some observers believe this lesion to be the end result of rheumatic infection which occurred in early life but was not severe enough to be recognized. Others regard it as an arteriosclerotic process, or the sequela of some early non-rheumatic infection.

An intense, rough systolic murmur at the base, a palpable thrill, and, in most cases, diminution or absence of aortic second sound are observed. Moderate enlargement is the rule A diastolic murmur is rarely heard, despite the presence of slight aortic insufficiency, noted *post mortem*. X-ray shows calcium deposits in the aortic valve region

Arteriosclerotic or Hypertensive Dilatation of Aorta. A systolic murmur heard best in the second right intercostal space is by no means peculiar to aortic stenosis Its most common cause is widening of the aorta due to arteriosclerosis or hypertension, less often to syphilis This murmur is blowing, not as harsh as in aortic stenosis, and not accompanied by thrill The aortic second sound is apt to be accentuated and have a ringing quality. Occasionally in marked ... [illegible] ...ic ascending aorta, dilata- ... [illegible] ...*lative* insufficiency of the ... [illegible] ... clinical features serve to establish correct diagnosis

Physiologic Basal Systolic Murmur. This murmur, usually heard in the pulmonic area, is short, usually soft and blowing, and apt to vary with respiration It does not mask either heart sound and is not accompanied by a thrill.

Anemia and Thyrotoxicosis. In severe anemia, a systolic or diastolic murmur, or both, and in severe thyrotoxicosis, a systolic murmur, may be heard in the aortic region, as well as elsewhere Until recovery from the underlying malady takes place, these murmurs may be indistinguishable from those of structural valvular disease With recovery, they disappear.

Aneurysm. Aneurysm of the ascending aorta or innominate artery may give every sign of aortic stenosis except the characteristic pulse and diminution of the aortic second sound, although the latter may occur if there is associated syphilis of the aortic valve With aneurysm there may be a well-marked tactile thrill and a loud systolic murmur transmitted to the neck, but one would probably also find abnormal pulsation, some difference between the radial pulses or the pupils, or indications of pressure on the trachea, a bronchus or recurrent laryngeal nerve Pain may be present X-ray establishes diagnosis.

Relative Pulmonic Insufficiency. Cases having marked increase of pulmonary vascular pressure may develop relative insufficiency of the pulmonic valve, characterized by a murmur indistinguishable from that due to early aortic insufficiency It is not common When associated with mitral stenosis it is known as the *Graham Steelle* murmur. Other cardiac findings establish diagnosis. The peripheral vascular signs of aortic valve regurgitation are rarely helpful because they occur only with free regurgitation, it is only the murmur of slight aortic insufficiency which might be confused with that of pulmonic insufficiency.

Pulmonic Stenosis. The rough systolic murmur and accompanying thrill are most intense to the left of the upper sternum If, as sometimes happens, the

murmur is best heard in the aortic area, it may be distinguished from that of aortic stenosis by the fact that it is not transmitted into the vessels of the neck and is not accompanied by the characteristic peripheral pulse changes X-ray and electrocardiograms show evidence of right-sided enlargement.

Patent Ductus Arteriosus. The murmur usually lasts through systole and diastole, is humming in character, may be accompanied by a thrill, and does not often affect the pulse wave.

Associated Mitral Disease. It is sometimes difficult to decide whether or not mitral disease is present in association with aortic disease, especially predominating regurgitation. A systolic murmur at the apex might be due either to structural mitral regurgitation or to relative insufficiency caused by left ventricular dilatation; a diastolic rumbling murmur, to either structural or relative mitral stenosis In all but the most severe grades of aortic regurgitation, a late diastolic rumble usually means structural mitral stenosis; in the severe grades it might mean either relative or structural stenosis Differentiation is often impossible However, because of the predominance of mitral stenosis in rheumatic heart disease, a questionable murmur, on the law of averages, would indicate structural mitral disease.

Tricuspid Disease

Regurgitation. As a rule, rheumatic disease of the tricuspid valve is not recognized, since it is usually associated with and masked by mitral disease Regurgitation can be *relative* or *structural*; the former results from dilatation and hypertrophy of the right ventricle secondary to disease on the left side of the heart, especially mitral stenosis. One finds evidence of pronounced congestion in the peripheral and portal circulations, marked dilatation of the right auricle, and a blowing systolic murmur heard best to the left of sternum in the region of the fifth costal cartilage The cervical veins are distended Systolic pulsation of the internal jugular veins, indicated by an outward movement of the overlying sternomastoid muscles, is evident In contrast to the undulating pulsation seen in the normal person or in one with congestive failure without tricuspid regurgitation, this movement is characterized by a pronounced systolic thrust as blood is regurgitated into the veins during systole and an abrupt collapse at the start of diastole To distinguish it from carotid artery pulsation, one can press gently against the jugular bulb, which lies behind the sternomastoid muscle just above the sternoclavicular junction, venous pulsation will disappear, arterial, not.

If systolic pulsation of the liver is present, the diagnosis of tricuspid regurgitation is further supported One must be careful to distinguish actual pulsation from systolic movement sometimes transmitted to the liver by the abdominal aorta or right ventricle. When it is actually pulsating, one can feel a definite expansile movement synchronous with heart beat if its lower part can be firmly grasped, or if one hand is placed anteriorly and the other posteriorly over the lower ribs on the right.

To differentiate clinically between relative and structural tricuspid regurgi-



FIG 16 26 Marked dilatation of cervical veins in rheumatic mitral disease Pronounced engorgement (and clinically observed deep pulsation) of veins provided support to the added diagnosis of tricuspid regurgitation (Courtesy Dr James H Currens)

tation is virtually impossible If, however, treatment of congestive failure or surgical relief of mitral stenosis is followed by diminution or disappearance of the systolic murmur and signs of peripheral and portal congestion described above, relative rather than structural impairment can be assumed

Stenosis. Tricuspid stenosis should be suspected if a diastolic rumble similar to that of mitral stenosis is heard over lower sternum or to right of midline, and if the right auricle is even larger than is usual with ordinary right-sided failure; the right cardiac border may be found well out toward the right. Cyanosis and venous engorgement are pronounced and the liver is apt to remain persistently enlarged. Orthopnea, dyspnea, and indications of pulmonary congestion are less striking than would be the case if the right-sided failure were solely a reflection of disease on the left side of the heart. In stenosis or regurgitation, enlargement of the right auricle with prominence of the superior vena cava and azygos vein may be observed by x-ray but are often hard to identify.

PULMONIC DISEASE

Regurgitation. When present in rheumatic infection, pulmonic valvulitis is usually an acute process which rarely leaves sufficient deformity to create a mechanical defect. As a rule, regurgitation is due, not to valvular disease, but

to dilatation of the ring based on increased tension in the lesser circulation It occurs in mitral stenosis, less often in left ventricular failure secondary to mitral insufficiency or aortic disease. One hears an early diastolic blowing murmur usually along the left sternal border, and increased pulmonic second sound The murmur cannot be distinguished from that of mild aortic regurgitation If the peripheral vascular signs of aortic regurgitation are present, they indicate that the aortic valve is the source of the murmur; their absence does not exclude aortic regurgitation. X-ray shows enlargement and increased pulsation of the pulmonary artery and right ventricle

Stenosis. This is rarely encountered in rheumatic disease. It is almost always a congenital lesion (*see* Chap. 15)

Combined Valvular Disease

Involvement of more than one valve occurs in approximately one-half of the cases of rheumatic heart disease, the usual combination being mitral disease with predominating aortic regurgitation.

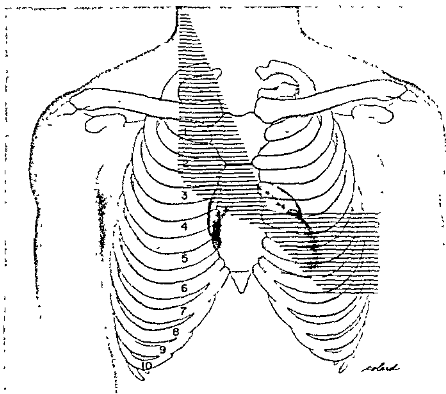


FIG 16 27 Systolic murmur in mitral disease with regurgitation and aortic disease with stenosis It is heard all over the precordium, loudest in aortic and mitral areas, faintest midway between them

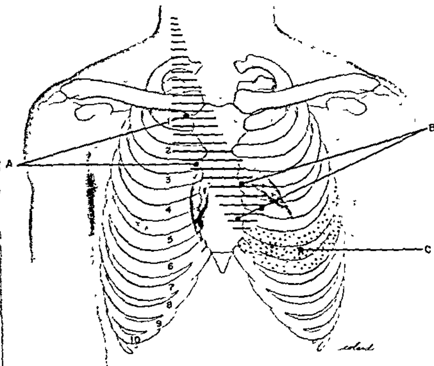


Fig 16 28 Distribution of murmurs in combined aortic disease with stenosis and regurgitation and mitral disease with stenosis and regurgitation

Careful study of the murmurs, especially their distribution, character, and effect on the heart sounds, will usually provide a correct diagnosis But difficulties sometimes arise from the fact that the number of murmurs audible is no indication of the number of valve lesions For example, in aortic disease, one might find the basal systolic and diastolic murmurs of its stenosis and insufficiency and, in addition, systolic and diastolic murmurs at the apex. The two latter, though usually indicative of structural mitral disease, could be caused by relative insufficiency and stenosis secondary to the left ventricular dilatation If the murmurs themselves do not provide conclusive evidence, there may be secondary signs substantiating the diagnosis of more than one lesion.

The signs of mitral disease combined with aortic regurgitation may not differ essentially from those of pure mitral disease, except that if both exist, enlargement of the heart is apt to be more general rather than limited to one side or the other. The manifestations of an aortic lesion can be distinctly

modified by associated mitral disease. If the latter is marked, left ventricular hypertrophy might be less and the peripheral pulse changes minimal

If mitral and tricuspid disease coexist, stasis in the peripheral and portal circulations and dilatation of the right auricle will be out of proportion to what would be expected on the basis of the degree of congestion found in the pulmonary system In any event, the diagnosis of tricuspid valvulitis, even in expert hands, is often open to question.

SYPHILITIC CARDIOVASCULAR DISEASE

Syphilis often involves the aorta, rarely, the myocardium or peripheral arteries. Although invasion of the cardiovascular system probably occurs early in acquired infection, 10 or more years usually elapse before clinical signs become evident Occasionally the interval is shorter. Cardiovascular involvement in congenital syphilis is rare

SYPHILITIC AORTITIS

The fundamental pathologic process is destruction of the media. Weakening of the wall and loss of elastic tissue result in dilatation of the vessel with wrinkling, thickening and irregularity of the intima Calcification may occur as a late manifestation Localized destruction or break of muscular or elastic tissue may result in circumscribed dilatation (*diffuse aneurysm*) or outpocketing (*saccular aneurysm*) Rupture may be an end result The ascending aorta is most often affected, less frequently, the transverse or descending portion; rarely, the abdominal When the process is in the ascending aorta, it often extends toward the heart, involving the aortic ring, cusps of the aortic valve, sinuses of Valsalva, and coronary orifices

Uncomplicated Syphilitic Aortitis

In the absence of complications, syphilitic aortitis causes no characteristic symptoms In fact, the disease cannot be diagnosed with certainty unless there is an appreciable degree of aortic valve involvement or an aneurysm It should be suspected if one finds dilatation without obvious cause or indications of impaired coronary circulation along with a history or other manifestation of syphilis

On physical examination one might hear a systolic, blowing murmur in the aortic area and perhaps some tympanitic, bell-like accentuation of aortic second sound X-ray will show prominence of ascending aorta and, fluoroscopically, especially in the left anterior oblique projection, increased amplitude of pulsation of the ascending segment. These variants cannot be regarded as significant unless found in a patient with a history of syphilis or a positive serologic reaction, and without hypertension or extensive arteriosclerosis, which are much more frequent causes

Syphilitic Aortitis with Aortic Regurgitation

This form of syphilitic cardiovascular disease produces most of the serious cases. When the valve is involved *the process of destruction and repair widens* the commissures between the cusps and, at the same time, the latter are pulled back toward the sinuses of Valsalva and anchored there. Regurgitation is the obvious result Stenosis does not occur The effect of syphilis is to widen the ring, in contrast to rheumatic or calcareous disease in which the pathologic process causes both stenosis and regurgitation.

Symptoms

Except for the possibility of vague substernal discomfort, no symptoms appear in the early stages Later when regurgitation has developed to the point of creating appreciable strain on the left ventricle, increasing dyspnea and eventually the usual picture of left ventricular failure will appear If, at any stage, the process impinges on a coronary orifice, angina pectoris may develop.

Early Signs

Murmurs. The first dependable sign of aortic valve involvement is the soft, blowing early diastolic murmur of slight aortic regurgitation It is best heard to the left of the sternum at the level of the third and fourth interspaces, less often at the lower end of the sternum, and occasionally at the cardiac apex or in the aortic area *In any case with a syphilitic history or in which syphilitic aortitis is a possibility, this murmur should be carefully listened for* with the *patient in various positions and with his lungs at the point of expiration.* When there is nothing to suggest rheumatic heart disease, especially if there is other evidence of syphilis, this murmur usually indicates syphilitic aortitis with beginning involvement of the valve One may hear a soft, blowing systolic murmur at the base, indicating dilatation of the aorta, by itself this has little significance because of its frequency in arteriosclerosis and hypertension.

Dilatation of Aorta. This can be detected by x-ray; physical signs are rarely dependable.

Later Signs

Murmurs. At the Base As progression of the disease causes further valvular insufficiency, the diastolic murmur increases in duration, intensity and area of distribution *The basal systolic murmur also becomes more intense and widespread* but remains blowing in character, never developing the harshness characteristic of aortic stenosis

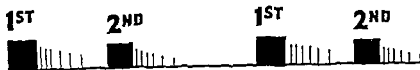


Fig 17.1. Sounds at base in early syphilitic aortitis with beginning aortic regurgitation Systolic murmur in aortic area Early diastolic murmur follows second sound, it is usually best heard along left border of sternum

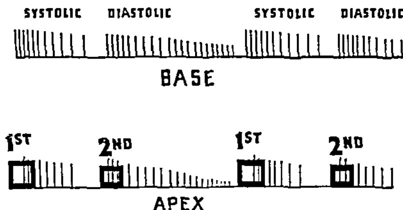


FIG 17 2 Sounds heard in advanced syphilitic aortitis with regurgitation At base, a loud systolic murmur masks or replaces first sound and a loud diastolic murmur replaces second sound At apex, a systolic murmur which may or may not mask first sound and a diastolic murmur which may or may not mask second sound

AT THE APEX. When left ventricular enlargement becomes pronounced, one will hear the systolic murmur of relative mitral insufficiency and sometimes the diastolic (*Austin Flint*) murmur of relative mitral stenosis. Both may be obscured by transmission of the basal murmurs.

Changes in Heart Sounds. Fixation of valve leaflets causes diminution and eventual absence of aortic second sound. The systolic murmur may mask or replace the first sound, especially at the base.

Cardiac Enlargement. Left ventricular enlargement results from the handicap imposed by the inefficient aortic valve Syphilitic aortitis is responsible for many of the largest hearts; in advanced cases, the cardiac impulse will be found well to the left of its usual position, in the sixth or sometimes in the seventh interspace, and often so forceful as to cause heaving of the chest wall

Peripheral Vascular Changes. Suprasternal and cervical pulsation, increased pulse pressure, and other peripheral vascular variants secondary to free aortic regurgitation are found in advanced cases (*see* Chap. 16).

X-Ray Findings

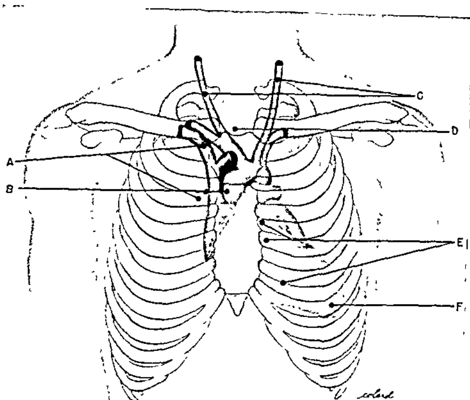
In addition to the changes observed in uncomplicated aortitis, calcium deposits may be evident in the aortic wall. When the regurgitation becomes well-established, the picture will be similar in other respects to that of rheumatic aortic regurgitation

Electrocardiographic Findings

If the leak is sufficient to impose appreciable strain on the left ventricle, evidence of left ventricular hypertrophy is found.

Additional Features

Two other aspects of syphilitic aortitis with aortic regurgitation must be emphasized:



A. SYSTOLIC MURMUR
B. PERHAPS DULNESS DUE TO DILATED AORTA
C PULSATING CAROTID ARTERIES
D PULSATION IN SUPRASTERNAL NOTCH
E DIASTOLIC MURMUR
F. APEX IMPULSE DISPLACED SYSTOLIC AND DIASTOLIC MURMURS

Fig 17 3 Important signs in advanced syphilitic aortitis with aortic regurgitation

1 The occurrence of partial or total obstruction of one or both coronary orifices by the inflammatory process, leading to angina pectoris and, sometimes, sudden death This can also occur in syphilitic aortitis without appreciable aortic regurgitation. The anginal attacks may be unrelated to effort and are often severe A syphilitic background should be seriously considered when attacks of angina pectoris occur in a young person, are atypical in character or predominantly nocturnal

2 The rapidity with which the patient loses ground, once the heart has begun to fail. Cardiac reserve can rarely be restored for more than a short period. In rheumatic valvular disease or congestive failure from other cause, proper therapy is more likely to be effective.

Diagnostic Pitfalls

Rheumatic Aortic Regurgitation. To distinguish between this and syphilitic aortic insufficiency is often difficult. The following points are helpful in the *early* stages:

1. History. Known rheumatic *infection in the past, or long-standing valvulitis points to rheumatic infection;* known syphilis suggests this disease as the cause.

2. Serologic reaction. As a rule, a positive test points toward syphilitic aortitis However, a patient with rheumatic heart disease could conceivably show a positive serologic reaction due to previous syphilitic infection without necessarily having syphilitic heart disease. On the other hand, in late cardiovascular syphilis the blood reaction is sometimes negative although since the introduction of the Kahn, Hinton, and other flocculation tests, this paradox is less likely than when only the Wassermann was available. The TPI test will be positive in an untreated case.

3 X-ray. Dilatation of the ascending aorta and, on fluoroscopy, increased pulsation favor syphilis.

In the *late* stages other helpful observations are:

1. Degree of aortic dilatation. This will be most pronounced in advanced syphilitic aortitis

2. Degree of regurgitation Regurgitation is most likely greater in syphilis than in rheumatic valvulitis which usually has some degree of stenosis and consequently less regurgitation The larger the heart and the more well-established the peripheral vascular signs, the greater the regurgitation

3. Character of the sounds and murmurs in the mitral area The presence of structural mitral stenosis, indicated by the characteristic diastolic murmur and a sharp or snapping first sound, would point toward rheumatic disease The Austin Flint murmur associated with free aortic regurgitation might be confused with that of true mitral stenosis, but here the first sound would lack the snapping quality, and the evidence would be in favor of syphilis.

4 Rapidity in progress of the disease After the signs of cardiac failure ap-



FIG 17 4 Syphilitic aortitis Dilatation of aorta, arrows point to rim of calcification in ascending segment and arch Left ventricle enlarged because of aortic regurgitation

pear, the downward trend in syphilitic disease is more rapid than in rheumatic disease

Calcareous Aortic Disease. Here there are signs of predominating stenosis which are not found in syphilitic aortic disease.

The features which differentiate syphilitic aortitis from arteriosclerotic or hypertensive dilatation of the aorta, relative pulmonic valve insufficiency, and changes due to anemia and thyrotoxicosis, are similar to those which distinguish these disorders from rheumatic heart disease (*see* Chap 16).

Syphilitic Aortitis with Diffuse Aneurysm

Probably because of the earlier recognition and improved treatment of syphilis, aneurysm is now encountered less frequently than it was formerly Aneurysm alone produces no cardiac embarrassment When symptoms attributable to the heart are present they are due to associated aortic regurgitation or coronary orifice obstruction Diffuse aneurysmal dilatation is discovered by x-ray, which shows a fusiform swelling of the aorta with increased, normal, or diminished pulsation It is of importance only because of the possibility of rupture

Syphilitic Aortitis with Saccular Aneurysm

Saccular aneurysm becomes evident clinically when it has enlarged sufficiently to press upon or invade a regional structure Earlier the diagnosis can be made only by x-ray as described in subsequent paragraphs

General Manifestations

When a thoracic saccular aneurysm is large enough to affect a nearby structure, one or more of the following indications may be present

Hoarseness or Aphonia. Pressure on the left recurrent laryngeal nerve is the cause.

Cough. This may reflect pressure on the trachea, a bronchus, or the recurrent laryngeal nerve. In the last named, it has a peculiar characteristic harsh quality described as *brassy*

Inequality of Pupils. One or the other contracts because of pressure on the homolateral cervical sympathetic nerve

Inequality of Brachial Blood Pressures. To be significant, the difference between the two arms must be 20 mm or more Inequality of radial pulses, being often congenital, is inconclusive

Stridor or Dyspnea. Interference with free flow of air through the trachea is the cause

Tracheal Displacement or Tug. (*See* Chap. 5)

total (*see Chap. 21*).

Manifestations Related to Site of Aneurysm

The clinical and x-ray findings which depend on the location of the lesion are as follows·



Fig 17 5 Bulging of chest wall due to aneurysm of ascending aorta.

Aneurysm of Ascending Aorta. This usually extends anteriorly and may reach the upper chest wall near the sternum Substernal pain is likely Dulness and sometimes visible pulsation may be found to the right of upper sternum. Pulsation is best detected by having the patient lie between the light and the observer, whose eyes should be level with the anterior thoracic wall. X-ray shows increased arc of the ascending aorta, best seen in the left anterior oblique projection In this position increased amplitude of pulsation may be detected fluoroscopically If calcification in this area is greater than that seen in the proximal descending aorta, syphilis is much more likely than arteriosclerosis This observation is peculiar to the ascending aorta; increased calcium in a lesion further along does not have the same implication

With continued enlargement, the aneurysm will eventually erode part of the sternum and adjacent second and third costal cartilages, appearing as a firm swelling under the skin. Palpation may reveal expansile pulsation or outward motion of the chest wall with each cardiac systole Abnormal pulsation in the suprasternal notch or in the neck is not a dependable sign; hypertension and [illegible] regurgitation are more common causes Murmurs and thrill may or m[illegible] no diagnostic significance

[illegible] aorta, this, as a rule, can be diagnosed [illegible] are dependent on pressure on the mediastinal structures, except [illegible] it may appear as a pulsating mass above the suprasternal notch Dilatation is best seen roentgenologically and fluoroscopically in the right anterior oblique projection. Pulsation is usually less apparent than when the ascending aorta is involved

Aneurysm of Descending Aorta. Usually extending posteriorly and perhaps



Fig 17 6 A Aneurysm of upper portion of descending aorta
B Lateral film showing barium in esophagus, which is dilated above point of obstruction caused by pressure from the aneurysm Erosion of vertebrae is also evident

eroding the spine, the most characteristic feature is severe, constant pain in the back, often radiating along the intercostal nerves. In the advanced case one may find signs of compression of part of the left lung and abnormal pulsation or swelling in the region of the left scapula. X-ray will show localized enlargement best seen in the left anterior oblique view; erosion of a vertebra or atelectasis of a portion of the left lung may be observed.

Aneurysm of Abdominal Aorta. Arteriosclerosis is a more likely cause Syphilitic aneurysm is rare It is marked by pain, often intense, probably in the upper abdomen, but sometimes in the flank or back. It favors one side, usually the left A rounded, pulsating mass may be palpated To be sure that one is not dealing with a dynamic aorta, or a solid tumor transmitting normal aortic pulsation, an expansile mass must be outlined by the palpating fingers By x-ray a circumscribed lesion is seen in the aortic region Calcium, if present, favors a diagnosis of aneurysm as opposed to tumor Erosion of a vertebra may be observed

Diagnostic Pitfalls

Suprasternal Pulsation. Aortic arteriosclerosis, hypertension, or the dynamic throbbing of a normal aortic arch often observed in a thin person, is a more likely cause than syphilis. In the first two, some widening of the supracardiac shadow by x-ray and a blowing systolic murmur at the base are common. Aneurysm can be diagnosed only when some of the indications described in the preceding paragraphs are present.

Aortic Stenosis. The murmur and thrill occasionally observed in aneurysm of the ascending aorta might be confused with those of aortic stenosis, especially

in older men Diminution of aortic second sound and the pulse and blood pressure changes characteristic of aortic stenosis are not present in aneurysm. X-ray is important.

Tumor. Differentiation between tumor and aneurysm is often difficult. In mediastinal tumor large enough to give signs at the surface, dulness is apt to extend to both sides of midline; in aneurysm it is usually unilateral Tumor often transmits impulses from the heart or aorta but the pulsation lacks the expansile character of aneurysm. If the tumor is malignant, its progression is more rapid than that of aneurysm; such indications as loss of weight and anemia will appear. Evidence of metastatic involvement of some other structure might be the key to diagnosis In some instances, differentiation between aneurysm and thoracic or abdominal tumor can be accomplished only by arteriograms.

Arteriosclerotic Aneurysm. Especially in the older age group, abdominal aneurysm is much more likely due to arteriosclerosis than to syphilis In the thorax the reverse is true, but arteriosclerotic aneurysm does occur. Tortuosity, elongation and generalized calcification of the aorta demonstrable by x-ray favor the diagnosis of arteriosclerosis.

SYPHILIS OF MYOCARDIUM

Gumma. This is not common, but may occur anywhere in the heart wall Indications are absent unless the lesion involves the conduction system, in which event signs of auriculoventricular or intraventricular block will appear.

Diffuse Syphilitic Myocarditis. Rare in this country, diffuse syphilitic myocarditis may account for some cases of heart failure not otherwise explainable The disease is said to be fairly frequent in communities where syphilis is particularly prevalent It cannot be recognized clinically but should be suspected in cases of cardiac insufficiency which are not due to other, more usual, causes.

SYPHILIS OF ARTERIES

Very rarely, syphilis involves the peripheral arterial system, producing inflammatory reactions which may result in thrombosis and occlusion and which are similar, clinically, to other types of endarteritis Occasionally, localized weakening of the wall of a small vessel will produce aneurysm.

18

HEART DISEASE IN RELATION TO OTHER INFECTIONS

ACUTE BACTERIAL ENDOCARDITIS

The *pneumococcus, hemolytic streptococcus, gonococcus, staphylococcus,* meningococcus, influenza bacillus and certain other organisms can invade the heart and create an acute fulminating illness lasting a few days to weeks The process is rarely an independent disease but occurs as one of the features of an overwhelming generalized infection The organism enters the blood stream from some focus, such as an infected wound, throat, or uterus, and reaches the heart in the course of its advance through the body. Pneumococcic carditis is an occasional complication of pneumococcus lobar pneumonia Gonococcic endocarditis sometimes follows a severe attack of gonorrhea, particularly if the prostate and seminal vesicles or the female pelvic organs are involved

How the heart is affected by any of the organisms mentioned above depends partly on the nature of the organism itself, partly on other factors which are not clearly understood The gonococcus attacks chiefly the endocardium In cases of pneumonia or hemolytic streptococcus infection, it is not unusual to find extensive serofibrinous or purulent pericarditis without any evidence of endocarditis Both may be present. Pneumococcus or staphylococcus endocarditis *often occurs without pericarditis In staphylococcus infection, there are frequently* miliary abscesses of the myocardium along with the endocarditis—a feature not common in the others Yet some degree of myocarditis is present with many infections, as will be indicated later

The characteristic lesion of acute bacterial endocarditis consists of one or more large vegetations, sometimes 0.5–1 cm in diameter or larger, composed of irregular masses of fibrin, platelets and bacteria The affected cusp may show extensive ulceration Any valve may be involved. *Unlike the subacute (Streptococcus viridans) type, acute bacterial endocarditis is just as apt to affect a normal valve as one which has been damaged previously.* Sometimes the vegetations are attached not only to the valve cusps but also to the chordae tendineae and, occasionally, even to the endocardium of a heart chamber

The diagnosis of acute bacterial endocarditis depends more on certain general phenomena than on any characteristic signs related to the heart Cardiac involvement is obscured by the indications of general infection. One sees the pic-

ture of acute septicemia, with marked prostration, high fever, and often recurrent chills. The discovery of any of the above-mentioned organisms in a blood culture, especially if they are found in large numbers, is good reason for suspecting bacterial endocarditis However, it is by no means pathognomonic, since most cases of septicemia uncomplicated by endocarditis will show one or more positive blood cultures. Occurrence of frequent septic emboli in the lungs or periphery is also suggestive, but here the picture is complicated by the fact, especially in the case of the lungs, that the emboli may be arising from a thrombophlebitic vein.

Significant signs in the heart may be lacking A murmur may be present as a result of interference with blood flow by a vegetation—a circumstance which can be definitely assumed if the murmur changes from day to day One which does not change must be interpreted with caution since it might well have been present prior to the illness, or be the result of dilatation based on myocardial weakness due to the toxemia Persistence of a murmur following recovery from septicemia may be taken as a presumptive indication of endocardial damage, provided one is sure that it did not exist prior to the acute illness.

Before the advent of antibiotics, bacterial endocarditis was almost always fatal With current therapy survival can be expected in a high percentage of cases but the patient may be left with cardiac impairment secondary to the valvulitis If antibiotic therapy is ineffective, death usually occurs from overwhelming infection

SUBACUTE BACTERIAL ENDOCARDITIS

Seen usually in late childhood or early adult life, subacute bacterial endocarditis is almost always caused by *Streptococcus viridans*, rarely by the gonococcus, influenza bacillus, or some other organism. It occurs where there has been previous valvulitis or at the site of a congenital anomaly, especially patent ductus arteriosus or interventricular septal defect. The susceptibility of the endocardium to invasion is not necessarily proportionate to the extent of the underlying lesion

As a rule only one valve is involved The disease is most likely to affect the mitral, probably because this valve is the most frequent site of rheumatic disease Vegetations composed of irregular masses of fibrin, blood cells, and bacteria are deposited on a leaflet They are much larger than the small excrescences seen in active rheumatic infection Sometimes they spread to the chordae tendineae, the wall of a heart chamber, or the aorta Ulceration through a septum or a valve leaflet occasionally occurs Focal myocardial lesions may be present but are of secondary importance

As in acute bacterial endocarditis, the diagnosis of the subacute form depends more on general manifestations than on cardiac findings but in a suspected case detection of valvulitis or a congenital anomaly lends important support The picture is one of steadily progressive subacute infection appearing in a patient with known rheumatic or congenital cardiovascular disease. Prior to the advent of antibiotic therapy, duration of several months, sometimes even 20 or

more, was not unusual, but today one rarely encounters such long-drawn-out cases. Onset is usually insidious. It may follow an acute upper respiratory infection, labor, or a surgical operation, especially removal of a tooth or the tonsils. Sometimes without any such preliminary episode, the patient simply begins to feel sick and shows evidence of non-localized *infection which at first does not* appear serious Fever, variable in degree, is usually present, but there are exceptional longer or shorter afebrile periods, especially during the stages when blood *cultures are negative*

Signs

As the disease progresses, one or more of the following manifestations will appear.

Increase of Fever. Associated chills and sweats are common.

Transient Joint Pains and Tenderness.

Progressive Anemia.

Petechial Hemorrhages. These appear, often in crops, as small reddish or purplish spots which do not disappear on pressure but fade out in a few days In a suspected case, they should be looked for daily on the skin—especially chest, abdomen, *forearms and hands—mucous membranes, conjunctivas and retinas* Under the nails they tend to be linear (*splinter hemorrhages*)

Embolism. Since the endocardial lesion is usually on the left side of the heart, embolism is more likely to occur in the brain, spleen, kidneys, extremities, or mesentery than in the lungs Embolism in the spleen is indicated by pain in the left upper quadrant and possibly splenic enlargement, in a kidney, by pain in the costovertebral angle and transient, microscopic hematuria; in the brain, by transient or permanent paresis or other indication of disturbed circulation, in the mesentery, by a picture resembling intestinal obstruction (*see* Chap 29)

Clubbing of Fingers and Toes. This is not as marked as in a congenital cardiovascular defect or chronic pulmonary disease.

The heart may show only the evidence of previously *existing valvular disease* or congenital defect indicative of ground fertile for superimposed infection Change of a murmur from day to day or week to week is significant; in uncomplicated rheumatic heart disease this occurs only with fairly rapid dilatation or certain changes in rhythm

If the disease does not respond to antibiotic therapy, death results from long-continued infection or embolic phenomena In rare instances, rupture of a valve leaflet with ensuing acute failure, or coronary block by a vegetation extending from the aortic valve, is the cause of death Since the development of antibiotic therapy, cases have been encountered in which the infection has been arrested but intractable congestive failure has resulted from increased valvular damage, or progressive renal disease comparable to chronic glomerular nephritis has developed.

The diagnosis of subacute bacterial endocarditis can be made with certainty only if, in a case at least presenting some of the features mentioned above, Strep-

tococcus viridans or, rarely, some other organism is found in blood culture. Repeated cultures may be necessary.

This disease must be distinguished from active rheumatic infection and other prolonged febrile illnesses such as tuberculosis, typhoid fever, brucellosis, and certain types of malignant disease.

DIPHTHERITIC CARDIOVASCULAR DISEASE

Membranous Stage

Overwhelming toxemia may cause peripheral circulatory failure similar to that seen in other forms of severe infection, with pallor, apathy, weakness, sweating, tachycardia, poor pulse, and diminished blood pressure. Restlessness, dyspnea, and perhaps cyanosis are often present; they are usually due, not to cardiac insufficiency, but to obstruction of the upper respiratory tract by membrane or edema.

Convalescent Stage

When the local lesion appears to be undergoing healing or has healed, serious and often fatal myocarditis or peripheral circulatory failure may develop, independently or together. These complications are more likely to occur in severe cases, especially those in which the diphtheric process has involved the nasopharynx or a cutaneous lesion Their frequency can be reduced by early and adequate treatment with antitoxin

Myocarditis

In severe diphtheria, this may appear in the first week; usually, however, it develops later, perhaps not until the fourth or fifth week The earlier it develops, the more likely it is to be fatal The conduction system, the musculature, or both may be involved.

Conduction System. When this is only slightly affected, damage may not be enough to cause clinical symptoms or signs The only evidence of trouble is provided by the electrocardiogram, which will show varying degrees of auriculoventricular or intraventricular block If damage is more severe, some change in rhythm—tachycardia, bradycardia or arrhythmia—will be observed clinically and electrocardiographically It may appear only intermittently It is likely to be accompanied by restlessness, pallor, sweating, epigastric pain, and vomiting. Death from heart block or ventricular fibrillation is possible.

Musculature An electrocardiogram may show flattening or inversion of T waves before any clinical manifestation is apparent The earliest clinical sign is often gallop rhythm Later, indications of cardiac dilatation—enlargement, murmurs not previously present, fall in blood pressure, and signs of passive congestion—may appear Tachycardia is the rule unless the rate is affected by changes in the conduction system Apprehension, restlessness, pallor, epigastric pain, and vomiting usually accompany this form of myocarditis also. If the pa-

tient recovers, the heart suffers no permanent damage, although convalescence is necessarily slow.

Occasionally electrocardiographic changes, particularly those associated with block, may persist, but clinically the patient continues well.

Peripheral Circulatory Failure

This is the most likely cause of death in the late stage of diphtheria; it is thought to be due to pooling of blood in the splanchnic vessels, brought about by postdiphtheric paralysis of the motor end plates of their parasympathetic nerves It is thus considered a manifestation of postdiphtheric polyneuropathy, and its appearance coincides with other paralyses, such as the palatal and oculomotor. Onset is often sudden, with apprehension, pallor, epigastric pain, and vomiting The skin becomes cold and clammy, the pulse thready, blood pressure falls, and other signs of peripheral circulatory failure develop The patient may die within a few hours or remain in the state of collapse for several days, then die suddenly or gradually recover

MISCELLANEOUS INFECTIONS

Various infections in addition to those described above—typhoid fever, typhus fever, Rocky Mountain spotted fever, yellow fever, influenza, poliomyelitis, sarcoid, coccidioidomycosis, and others—may cause diffuse or focal inflammation in the myocardium As a rule, the damage is not clinically evident but may be indicated by non-specific ST-T changes on the electrocardiogram. In a serious case, circulatory failure may occur, it is more likely to be on a peripheral than a myocardial basis

Tuberculous pericarditis is a not infrequent complication of tuberculous disease elsewhere, especially in a lung, pleura, or mediastinal lymphnode Lesions may be found in the myocardium but are presumably of secondary import

Foci of infection in other parts of the body, such as chronic cholecystitis or sinusitis, may perhaps contribute to the severity of already existing heart disease, particularly to congestive failure or angina pectoris There is no conclusive evidence of such a relationship

ACUTE BENIGN PERICARDITIS

This disease, thought to be caused by a virus, has been frequently encountered during the past few years For the reasons indicated below it may be misnamed. Usually following an upper respiratory infection, it is characterized by acutely developing substernal or precordial pain, dyspnea, cough, fever, and often prostration Pericardial friction rub is almost always present, the heart area is increased in size, often greatly so. This change may be due to effusion but many observers believe that dilatation secondary to associated myocarditis is the predominating factor. The electrocardiogram shows the changes of acute pericarditis (*see* Chap. 23) Acute pleuritis is common, signs of trouble in the lungs may be found by physical and x-ray examination. Recovery with no evidence of permanent damage is the rule.



FIG 18 1 Acute benign pericarditis Patient developed clinical signs of fibrinous pericarditis a few days after onset of acute respiratory infection Cardiac shadow slightly widened Hazy density in right lower lung field due to associated pulmonary infiltration Clinical recovery and normal x-ray after 3 weeks

The enlargement of the cardiac area which is often not entirely attributable to pericardial fluid, the frequent involvement of pleura and lungs, and the pathologic findings in the few cases which have proved fatal, appear to indicate that this disturbance is not pericarditis alone; it should be regarded as a generalized infection of which pericarditis and often myocarditis are striking manifestations.

Rarely, a picture simulating acute benign pericarditis is encountered as a complication of mononucleosis.

19

HYPERTENSIVE HEART DISEASE

SYSTEMIC HYPERTENSION

Hypertension may occur in the peripheral vascular system (systemic hypertension), in the pulmonary vascular system (*pulmonary hypertension*), or in both Either type may cause heart disease The term *hypertensive heart disease* is applied to cases which show evidence of left ventricular hypertrophy or strain caused by peripheral hypertension *Pulmonary heart disease* or *cor pulmonale* refers to cases in which the right ventricle shows evidence of enlargement or strain as a result of pulmonary hypertension secondary to disease of *the lungs or pulmonary arterial tree*

Hypertensive heart disease is common Ninety per cent of cases are accounted for by essential hypertension, the remainder chiefly by chronic nephritis or chronic destructive renal lesions There are, as indicated in Chapter 9, many other causes, but in these the elevation of blood pressure is of secondary importance In any case of high blood pressure, with or without heart disease, care must be taken to determine its underlying cause, for if this can be removed *by proper treatment, cardiac or other complications may be avoided or arrested*

The exact nature of the relationship between hypertension and hypertensive heart disease has never been clearly understood It is possible that the heart disease is caused not by hypertension alone but by other, contributory factors; it may be that the same factors are causing both the hypertension and the heart disease *Such a hypothesis would help explain the fact that certain patients with* pronounced hyperpiesia live for years without showing evidence of cardiac impairment, while others with less elevation show it much sooner

Symptoms

Although hypertension may give generalized symptoms, such as chronic fatigue, irritability, headache, or vague gastro-intestinal complaints, it is often asymptomatic and *discovered accidentally There may be no subjective indications of hypertensive heart disease until failure sets in*

Signs

The following signs, if encountered in a patient who has high blood pressure and no other form of heart disease, *indicate hypertensive heart disease.*

Cardiac Enlargement. This is variable, depending partly on the degree and

duration of the hypertension and perhaps partly on other factors not understood Generally, but not necessarily, the greater the hypertension and the longer its duration, the more the enlargement. It is of the left ventricular type, the apex impulse, except in the lesser degrees, being forcible and below and to the left of its normal position *

Murmurs. When left ventricular enlargement is well-established, one will hear a soft or loud blowing systolic murmur at the apex based on relative mitral insufficiency. Dilatation of the aorta will cause a blowing systolic murmur in the aortic area

Increased Aortic Second Sound. In well-established hypertension the sound may be loud and ringing, in failure it may be overshadowed by increased pulmonary second sound due to secondarily elevated pulmonic hypertension

Pulsations in Neck. These may appear above or to the right of the sternal notch due to increased pulsation of the innominate artery, or over the carotid arteries The former may be sufficiently pronounced to suggest aneurysm.

X-Ray Findings

At first x-ray shows prominence of the left ventricular contour with perhaps some elongation and tortuosity of the aorta. Later, left ventricular enlargement becomes definitely evident and the aortic changes are more pronounced Eventually the picture will be that of generalized cardiac enlargement and failure (*see* Chap 14)

Electrocardiographic Findings

The picture is that of left ventricular hypertrophy.

Failure

If the patient escapes some fatal complication such as cerebral hemorrhage, renal failure, or an intercurrent disease, he will eventually develop cardiac failure To forsee the exact time of its occurrence on the basis of blood pressure level or its duration is impossible Broadly speaking, *a consistently high diastolic level is of more serious import,* for to maintain elevated pressure throughout the cardiac cycle imposes more strain on the heart than to raise pressure during systole alone The patient who shows wide fluctuations is likely to have a more benign form of the disease than one whose level remains high and relatively fixed

Tachycardia, gallop rhythm, pulsus alternans, or a loud pulmonic second sound may herald impending trouble As a rule, the earliest symptom of failure is progressive dyspnea, harassing cough, cardiac asthma, or attacks of anginal pain based on a combination of associated coronary arteriosclerosis and the increased blood requirement of the hypertrophied ventricular musculature With time the other signs of left ventricular failure, and eventually right ventricular failure, will develop (*see* Chap 14)

Before failure appears, the diagnosis of hypertensive heart disease usually presents no difficulties After it is well-established, diagnosis may be difficult

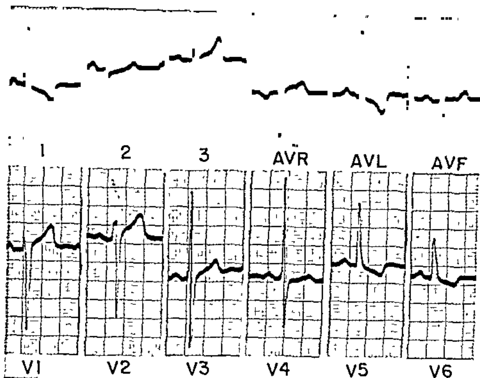


FIG 19 1 Left ventricular hypertrophy due to well-established systemic hypertension

unless hypertension is known to have existed previously The chief reasons are.

1. *When the heart fails, the systolic and diastolic pressures may fall appreciably*, so that hypertension as a cause cannot be proved. Furthermore, when a case of congestive failure is treated some of the very measures which help the heart *such as weight reduction and low sodium intake may also effect reduction of* blood pressure, in which event one cannot be sure of previous hypertension

2. The murmurs caused by cardiac dilatation might be wrongly interpreted as due *to structual valvular disease.* Whether failure is due to hypertension, rheumatic valvular disease, or coronary disease, sometimes cannot be determined by physical signs In such a dilemma one is forced to rely on the age of the pa*tient, a history taken with special reference to the previous existence* of syphilis, rheumatic infection or hypertension, and evidence of any of these diseases elsewhere in the body.

PULMONARY HYPERTENSION

Just as systemic hypertension, by imposing extra strain, eventually leads to failure of the left ventricle, so increased pressure in the pulmonary circulation will lead to failure of the right ventricle. Pressure in the pulmonary circuit cannot be measured by ordinary means but where evaluation is especially important cardiac catheterization may be employed. In the following disturbances pulmonary hypertension can be assumed to exist and eventually to be capable of causing, on the right side of the heart, changes comparable to those produced on the left by peripheral hypertension:

1. Failure of left ventricle from any cause
2. Mitral disease with predominating stenosis.
3. Certain congenital cardiovascular defects, especially patency of ductus arteriosus and wide auricular septal defect
4 Chronic bronchopulmonary disease.
5. Impairment of blood flow through the pulmonary artery or its branches

The mechanism by which pulmonary hypertension and eventually right ventricular failure is produced in cases of left ventricular failure, mitral stenosis and congenital defects has already been discussed (*see* Chaps 14 and 15)

Acute Cor Pulmonale

Sudden embolic obstruction of the pulmonary artery or one of its large branches causes acute strain on the right side of the heart, resulting in severe dyspnea, a sensation of suffocation in the chest, cyanosis, distension of neck veins and perhaps signs of shock (*see* Chap 27)

Chronic Cor Pulmonale

This is often but not necessarily found in any disturbance creating impairment of normal pulmonary ventilation such as hypertrophic emphysema, chronic fibrosing bronchopulmonary disease from any cause (chronic tuberculosis, silicosis, severe chronic bronchitis and bronchiectasis), intractable asthma, tracheal stenosis and severe thoracic deformity It also may occur when blood flow through the pulmonary artery or its branches is hampered by such a cause as thrombosis of a major artery following non-fatal massive embolism, multiple small emboli or thromboses, infiltration of arterioles by metastatic malignant disease, or pulmonary arterial and arteriolar endarteritis (*Ayerza's disease*)

Symptoms

As in the case of systemic hypertension, there are no symptoms directly attributable to heart disease until failure sets in Dyspnea and cough may be present but in the early stages they are due, not to the cardiac, but to the underlying pulmonary or vascular disturbance Later, when failure develops, these symptoms become more severe and are attributable to both cardiac and pulmonary changes

Signs

Signs may be absent until the appearance of failure. There may, on the other hand, be some of the following indications of cardiac impairment

Cyanosis. In the early stages this is more the result of pulmonary than of heart disease

Clubbing of Fingers and Toes. What has just been said about cyanosis applies here as well.

Right Ventricular Enlargement. One must depend on x-ray and electrocardiogram Physical examination is rarely helpful because of position of the right ventricle and because pulmonary emphysema or thoracic deformity may hamper percussion.

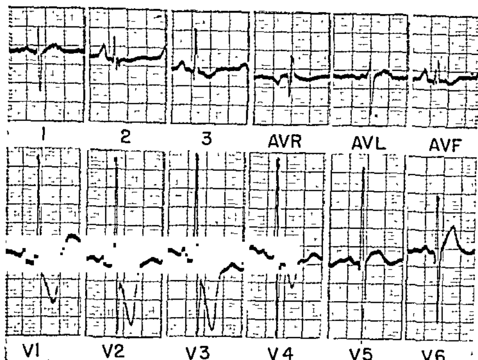


FIG 19 2 Pronounced right ventricular hypertrophy in a case of chronic cor pulmonale secondary to severe kyphosis with deformity of thoracic cage

Accentuation of Pulmonic Second Sound. Actually this is a sign of pulmonic hypertension, not of trouble *on the right side of the heart*

Elevation of Venous Pressure. One looks for distention and abnormal pulsation of jugular veins, and other signs described in Chapter 8

Right Auricular Enlargement. In the earlier stages this is detectable only by x-ray; later when it is pronounced, an area of dulness will be found to the right of the sternum.

X-Ray Findings

Initially one will observe engorgement of the pulmonary artery and its branches. Later right ventricular enlargement will be observed in the lateral projection as an encroachment of the cardiac shadow into the retrosternal space Right auricular enlargement appears as extension of the heart shadow to the right of the sternum Eventually the picture will be that of right-sided failure (*see* Chap 14).

Electrocardiographic Findings

The changes are those of right ventricular hypertrophy.

Before extreme enlargement or failure appear, pulmonary heart disease cannot be diagnosed on the basis of positive cardiac findings, although the latter may, when they are observed, substantiate the diagnosis. Its presence is inferred

when emphysema or some other bronchopulmonary disorder capable of causing pulmonary heart disease is present and there is no disease on the left side of the heart to account for the clinical picture. Another suggestive sign is compensatory elevation of hemoglobin and red blood count Some obstructive disturbance of the pulmonary arterial tree is to be suspected when cyanosis, elevation of hemoglobin and red count, and later, evidence of right-sided strain, appear in the absence of demonstrable chronic pulmonary disease, congenital cardiovascular defect, or disease of the left side of the heart

When the heart can no longer carry the added load the signs of right ventricular failure appear. jugular venous pulsation in the upright position, hydrothorax, enlargement of the liver, ascites, peripheral edema, marked cyanosis, and possibly diminution of pulmonic second sound

20

CORONARY HEART DISEASE

Heart disease due to coronary atherosclerosis is frequent in and beyond middle life, but it is often encountered in younger adults as well. It is more common in males than in females Progressive diffuse or localized narrowing reduces blood flow through the arteries thus creating an imbalance between the requirements of the myocardium and its supply. Thrombosis, subintimal hemorrhage, or hematoma *secondary to an atherosclerotic lesion causes acute obstruction.* Although the term coronary heart disease as ordinarily used refers to the arteriosclerotic process, comparable changes are occasionally seen in other diseases such as acute rheumatic infection, thromboangiitis obliterans, and polyarteritis nodosa

It was formerly thought that any appreciable degree of narrowing would reduce coronary blood flow enough to produce cardiac impairment But it is now generally accepted that extensive narrowing or obstruction even of one or more major coronary arteries may exist without seriously handicapping the heart. An area of myocardium deprived of its normal supply may receive blood through anastomotic vessels reaching it from the same major artery proximal to the point of obstruction or from some nearby unaffected artery. If the basic arterial disease progresses slowly enough to allow time for establishment of adequate collateral circulation, sufficient blood may be channeled into the affected areas to meet ordinary needs for years If collateral circulation cannot keep pace with the arterial changes or some added circumstance further disturbs balance between supply and demand, serious trouble results.

Angina pectoris is often the first and only clinical manifestation Or, coronary artery disease may be indicated by gradually increasing left ventricular dilatation and hypertrophy, dyspnea interfering with activity, and eventual progression to congestive failure. This chain of events results from *myocardial fibrosis* secondary to diminished blood supply Concomitant angina pectoris may or may not appear But as the *arterial disease advances, the likelihood of angina* pectoris, coronary insufficiency, or myocardial infarction becomes progressively greater. *Sometimes, acute obstruction with myocardial infarction is the* first sign of trouble.

The *diagnosis of uncomplicated coronary disease* is made on the basis of the history, often but not necessarily supported by electrocardiographic findings *There are no confirmatory* physical signs In many cases, the electrocardiogram

is normal. In others it shows non-specific T wave abnormalities or conduction defects due to impaired coronary circulation.

ANGINA PECTORIS

Angina pectoris is a syndrome of paroxysmal discomfort, gripping pain, or smothering sensation experienced in the chest. When mild it may be described not as pain but rather as a feeling of fullness, indigestion, gas, constriction, choking or temporary inability to catch the breath. Whatever form it takes, it is almost always experienced beneath the sternum, usually its upper or mid-portion, rarely the lower. Characteristically, it is precipitated by exertion, emotional stress, exposure to cold or any other circumstance which creates a need for increased cardiac work. In the advanced case, attacks may occur with the patient at rest in the absence of obvious provocation (*angina pectoris decubitus*). An episode begins suddenly, quickly reaches a maximum lasting 1 to 3 minutes or somewhat longer, and subsides gradually It often ends with a spell of belching probably due to accompanying reflex cardiospasm. Its severity may give the patient the mistaken impression of 5 to 15 minutes' duration The pain is likely to spread to the left shoulder or arm, following, in the latter, the course of the ulnar nerve. Sometimes the path is to the right or to both arms. Extension to the neck, jaw or throat, especially on the left, or to the epigastrium is occasionally encountered In rare instances, an attack will originate in one of these zones, it may be confined there but is more likely to spread to the substernal region. Relief is obtained by reducing or stopping activity and by the immediate use of nitroglycerine, a nitrite, or other coronary vasodilator.

Both angina pectoris and coronary insufficiency (*see below*) are presumably due to lack of enough oxygen in the myocardium to meet the needs of the moment. Inadequacy of blood supply, which may be relative, actual, or both, is brought about by one or more of the following causes:

1 Increased work load, as in unaccustomed severe exertion, emotional stress, exposure to cold, ingestion of a large meal, straining, paroxysmal rapid cardiac rhythm, hypertension, or thyrotoxicosis

2. Decreased volume flow through the arteries resulting from.
   a. Coronary artery disease, almost always atherosclerosis, very rarely some other form of arteritis, as in rheumatic infection, thromboangiitis obliterans, or polyarteritis nodosa.
   b. Coronary orifice obstruction, usually by an inflammatory lesion in syphilitis aortitis, very rarely by a vegetation in bacterial endocarditis
   c. Valvular disease, especially free aortic regurgitation, or advanced aortic mitral stenosis
   d Diminished cardiac intake from any cause, especially shock, hemorrhage, acute hypotension, or reflex insult to the circulatory apparatus, as by pulmonary embolism or some acute abdominal disturbance

3 Deficient oxygenation of blood as in anesthesia, pulmonary embolism, anemia, or impaired intrapulmonary exchange

It should be emphasized that more than one of these factors is usually operative For example, exertion which would not cause trouble in a person with normal coronary vessels may well precipitate an attack of angina in one with an appreciable degree of coronary atherosclerosis. In free aortic regurgitation angina may appear only when exertion or excitement increases the cardiac load. Bouts of distressing anginal pain in a patient with anemia will often disappear when the blood is restored to the normal level even though some coronary atherosis is present

The diagnosis of angina pectoris is made on the basis of the patient's description of his discomfort and its relation to the precipitating factors mentioned above, especially exertion and emotional stress. There are no pathognomonic physical signs. One may detect neither cardiac enlargement, murmurs, nor irregularity, although when some underlying disease such as hypertension or aortic regurgitation exists, its characteristic signs will be found. Pallor, sweating *and a distressed expression may be noted during an attack but these, of course, can be provoked by other forms of pain* Pulse and blood pressure may remain unaltered, either or both may show temporary elevation. There are no specific incriminating laboratory findings and x-ray will be normal or show only enlargement

The electrocardiogram may or may not be helpful Between attacks, many cases will show a normal electrocardiogram. Others, as indicated above, will *show non-specific T wave abnormalities or conduction defects which reflect poor* coronary circulation A tracing taken during an attack may remain unchanged or show ST segment depression of a flattened or downward sloping variety in several of the limb leads and in the lateral precordial leads (V4–V6), with or without associated T wave lowering or inversion Similar deviations may be transiently present immediately after an exercise or anoxemia test, even though pain is not precipitated

A careful history, correctly evaluated, is necessary to differentiate angina pectoris from the discomfort encountered in apprehensive states, neurocirculatory asthenia, and premature beats In the first two, the patient experiences an ache, short stabs of pain, or hyperesthesia of skin and ribs, not substernally, but over the precordium or at the apex. There is no feeling of constriction or compression In premature beats, *careful questioning will bring out that the* sensation is one of the heart "stopping" or "turning over." Another common and difficult problem is the differentiation of angina pectoris from cardiospasm associated with hiatus hernia or other cause. Pain due to disease in sites remote from the heart such as cholecystitis, subdeltoid bursitis, and chronic arthritis of the cervical or thoracic spine may also be confused with angina pectoris. Here not only a detailed history is required, but also a careful appraisal of the *findings obtained by all methods of examination.*

Since coronary atherosclerosis greatly exceeds all other disturbances as a cause of angina pectoris, this diagnosis is most likely to be correct once the presence of angina pectoris has been established However, especially in younger persons, it is highly important to exclude the other possibilities listed above.

CORONARY INSUFFICIENCY

The term acute coronary insufficiency or acute coronary failure is applied to episodes of pain similar to angina pectoris but more severe and prolonged, lasting from 10 or 15 minutes to several hours. The mechanism of their production is similar to that of angina Because of the longer duration of the myocardial ischemia, small patches of myocardial necrosis may develop, most likely in the subendocardial zone The pain is less amenable to the vasodilators. Mild shock, slight fever and leukocytosis are possible The electrocardiogram usually shows T wave inversion with slight elevation of ST segment in several leads. These changes which sometimes resemble those of acute pericarditis last from hours to days As in angina pectoris, the pattern will revert to that which existed prior to the episode, that is, normal or showing evidence of previous damage.

An attack of coronary insufficiency can be precipitated by exertion or other circumstance requiring increased cardiac effort or by diminution of blood flow from one of the causes noted above Recovery without evidence of added damage to the heart may occur, but if ischemia persists, definite myocardial infarction may result

Coronary insufficiency is distinguished from angina pectoris by the greater severity and duration of the pain, the comparative infrequency of attacks and, more often than not, transient electrocardiographic changes Myocardial infarction, on the other hand, is likely to show one or more of the following features, the severity depending on the extent of the damage: pain even more severe and prolonged, shock, fever, nausea and vomiting, alteration in heart sounds such as tic-tac or gallop rhythm, pericardial friction rub, leukocytosis, elevated sedimentation rate, and characteristic progressive changes in serial electrocardiograms Sometimes one cannot be sure whether he is dealing with coronary insufficiency or a relatively small infarction.

MYOCARDIAL INFARCTION

This common, dangerous, and often fatal complication may appear in a person with previous angina pectoris or known coronary arterial disease, or strike without warning one who has been regarded as perfectly well It can be brought about in one of three ways The first two causes noted below account for practically all of the cases; the third is excessively rare

1. Prolonged myocardial ischemia in the absence of a fresh occlusion when work load is increased or blood flow diminished.

2. Coronary artery occlusion due to fresh thrombosis or subintimal hemorrhage. Closure may occur either in the vessel which normally supplies the affected area or in a distant vessel which has, by collateral circulation, become its source of supply. The episode is likely to begin when the patient is at rest.

3 Embolism of a coronary vessel secondary to thrombosis formation in a left heart chamber, as in subacute bacterial endocarditis or mitral stenosis with auricular fibrillation.

Symptoms

Pain. This is almost always the first and outstanding symptom. If only a small segment of muscle is infarcted the pain may be no more than that encountered in an attack of angina pectoris or coronary insufficiency. Typically, however, because a larger segment of myocardium is deprived of its blood supply, the patient will experience a sense of *agonizing compression or crushing* most severe in the middle of the chest and behind the sternum. It often spreads throughout the thorax, sometimes into the upper abdomen, into one or both shoulders and arms or into the upper midback, face or neck. In a few instances it will be felt not substernally but solely in one of these other areas. It is accompanied by dyspnea, a sense of smothering, and collapse. Duration is usually proportionate to the extent of the insult. If a small area is involved, the pain may last only an hour or so, with a large infarction, it may persist for two or three days, although subdued after the first few hours. A few cases occur without distressing pain. The patient may note an attack of dyspnea or have no symptoms. Sometimes the development of acute pulmonary edema is the initial indication.

Dyspnea. This accompanies the pain; occasionally it is the predominating symptom.

Nausea and Vomiting. These are likely in serious cases, but are often attributable not to the disease but to morphine or other drug administered for the pain.

Signs

Peripheral Circulatory Failure. Except in mild cases, the patient presents the picture of shock, its severity depending on the degree of insult. The expression is drawn and anxious, the skin, especially of the face, shows a grayish, cyanotic tint and feels cold and clammy. The pulse rate is usually rapid, occasionally, because of heart block secondary to the infarction, it is slow. Blood pressure may rise initially because of pain but soon both blood and pulse pressures fall.

Cardiac Changes. Tic-tac or gallop rhythm, some abnormality of the beat, such as premature contractions, ventricular paroxysmal tachycardia, auricular fibrillation, or heart block, or evidence of cardiac dilatation may appear soon after onset or later.

Indications of Myocardial Necrosis. These develop a few hours or longer after onset. They are important aids in distinguishing myocardial infarction from lesser grades of coronary disease. The most important are:

Fever. Moderate elevation of temperature usually occurs within 24–48 hours after onset and lasts for two or three days. In the most severe cases, it may reach 103°–104° and require more time to subside. Generally speaking, the higher and longer the fever, the more severe is the infarction.

Leukocytosis. The leukocyte count is usually elevated. The greater the elevation and the longer it lasts, the graver the outlook.

Increased Sedimentation Rate. This is proportionate to the severity of the

attack The rate may remain rapid for several months or may never return to normal

Pericardial Friction. Two or three days after onset a pericardial friction sound, due to a patch of sterile pericarditis overlying the segment of infarcted muscle, may appear; it is usually best heard *to the left of the sternum in the* second, third or fourth interspace. The sign is confirmatory evidence of myocardial infarction and since it is often transient should be carefully searched for daily for several days following the onset of a suspected attack. Its absence does not disprove the diagnosis

STAGE	CHARACTERISTIC EFFECTS (Leads 1, AVL, V-1 to V-6)	RECIPROCAL EFFECTS (Leads 2, 3, AVF)
Early stage (hours to days)	ST segments marked elevation and upward coving T waves: beginning inversion Q or QS waves appear	ST segments depressed T waves early peaking
Developmental T wave stage (weeks to 1–2 months)	ST segments. returned almost to isoelectric line T waves deeply inverted Q or QS waves persist	ST segments isoelectric T waves further peaking
Receding T wave stage (months to years)	ST segments isoelectric T waves: slightly inverted, diphasic, or upright and low Q or QS waves: persist	ST segments isoelectric T waves smaller and normally rounded

Fig 20 2 Typical electrocardiographic changes in anterior myocardial infarction (Prepared with assistance of Dr Daniel Holzman)

STAGE	CHARACTERISTIC EFFECTS (Leads 2, 3, AVF)	RECIPROCAL EFFECTS (Leads 1, AVL, V-1 to V-6)
Early stage (hours to days)	ST segments marked elevation and upward coving T waves beginning inversion Q or QS waves appear	ST segments depressed T waves: early peaking
Developmental T wave stage (weeks to 1–2 months)	ST segments returned almost to isoelectric line T waves: deeply inverted Q or QS waves. persist	ST segments isoelectric T waves further peaking
Receding T wave stage (months to years)	ST segments isoelectric T waves slightly inverted, diphasic, or upright and low Q or QS waves: persist	ST segments isoelectric T waves: smaller and normally rounded

Fig 20 3 Typical electrocardiographic changes in posterior myocardial infarction (Prepared with assistance of Dr Daniel Holzman)

Embolism. Thrombus formation within the left ventricle secondary to endocardial damage in the infarcted segment may give rise to embolism of the brain, mesentery, an extremity, or other structure The episode usually occurs a week or more following onset Pulmonary embolism may occur if the right ventricle is involved. A more likely cause of pulmonary embolus, however, irrespective of the site of infarction, is a peripheral vein which has become the site of phlebothrombosis as a result of immobilization of the patient and stagnation of venous blood flow

Electrocardiographic Findings

Following acute myocardial infarction, serial tracings will show the changes indicated in Figures 20 2 and 20 3 They are almost certain to occur when the

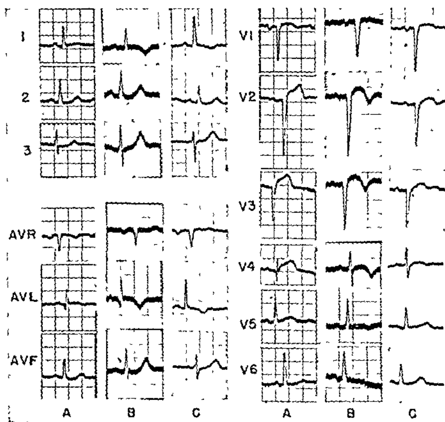


FIG 20 4 Anterior myocardial infarction Serial electrocardiograms showing sequence of changes

A Day of onset Q wave in AVL Absent R with Q S pattern in V1–V3 and Q in V4 ST segment elevated in 1, AVL and V1–V4

B Ten days after onset Q-S pattern essentially the same ST segments returning toward isoelectric line Inversion of T waves in 1, AVL, V1–V5 Reciprocal peaking of T waves in 2, 3 and AVF

C Two months after onset Q-S pattern persists in V1–V3 Further return of ST segments toward isoelectric line T waves remain inverted in 1, AVL and V1–V4 Reciprocal T wave peaking less pronounced

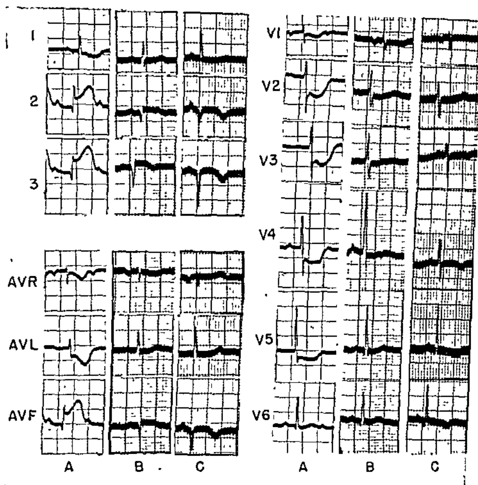


FIG 20 5 Posterior myocardial infarction Serial electrocardiograms showing sequence of changes
A Day of onset ST segment markedly elevated in 2, 3 and AVF Reciprocal depression of ST in 1, AVL, V1–V5
B Four days after onset Deep Q with diminution of R wave in 2, 3 and AVF ST changes less striking but still evident
C Three weeks after onset Q-S pattern with disappearance of R in 2, 3 and AVF Definitely inverted T waves in 2, 3 and AVF Inversion of T waves in V3–V6 is not characteristic of pure posterior infarction, it suggests that the area of ischemia also involves antero-lateral wall of left ventricle

infarct involves the total thickness of the ventricular wall. In a few cases, especially if the infarcted area is not completely transmural, non-specific ST-T deviations will be found The term anterior infarction refers to the anterior surface of the left ventricle, posterior infarction to its posterior surface The right ventricle or auricles become involved only by extension of the process from the left ventricle Anterior or posterior infarction may reach into the interventricular septum or the lateral wall of the left ventricle. Involvement may be detected on the electrocardiogram as extension of the typical changes of anterior or posterior infarction to include the precordial leads which reflect activity in the region affected. Serious trouble in the septum may be further in-

dicated by a permanent or temporary disturbance of conduction, most likely auriculoventricular block or bundle branch block.

The initial electrocardiographic changes created by myocardial infarction usually appear within a few hours and progress according to a fairly definite pattern so that it is often possible to estimate the age of the lesion If the clinical picture is suggestive of infarction but the diagnosis is not electrocardiographically substantiated in the early stages, serial tracings are imperative, since the changes sometimes appear tardily.

Diagnostic Pitfalls

Acute disturbances especially apt to be confused with myocardial infarction, particularly in the first few hours, are: coronary insufficiency, pulmonary embolism, acute pericarditis, dissecting aneurysm of the aorta, spontaneous pneumothorax, hiatus hernia, acute diaphragmatic pleuritis, and certain intra-abdominal insults, such as acute pancreatitis, acute cholecystitis, and perforation of a peptic ulcer. In such cases, very careful evaluation with all pertinent investigative procedures is often necessary to establish correct diagnosis. The appearance of typical changes in serial electrocardiograms may, in the case of coronary insufficiency, myocardial infarction, or pulmonary embolism, provide the answer before it becomes otherwise apparent

Death may immediately follow the acute episode or occur after a few days from congestive failure, ventricular fibrillation, pulmonary embolism, further myocardial infarction, or myocardial rupture. The patient may recover and live for years without having any further cardiac difficulty or he may develop chronic congestive failure, or subsequent attacks of angina pectoris or myocardial infarction—any of which may be fatal In rare instances, an aneurysm of the ventricular wall develops in the infarcted area, this does not influence the prognosis.

21

DISTURBANCES OF CARDIAC RHYTHM

A thorough understanding of the mechanism of the heart beat and the ways in which it can be disturbed is necessary for diagnosis, treatment and prognosis of abnormal rhythms Some are harmless, others serious Differentiation is essential, first because therapeutic requirements of the different types vary, and second because interpreting a minor disturbance as important may needlessly alarm the patient and lead to cardiac neurosis Since any aberrant rhythm is likely to be felt by the patient, he can easily become apprehensive

The impulse which starts the normal heart beat begins at the sinoauricular (S-A) node and spreads through the auricular musculature, causing it to contract From the auricles, it extends to the auriculoventricular (A-V) node, then along the bundle of His and its branches to the Purkinje fibers, through which it is distributed to the ventricular musculature Since the impulse reaches the two ventricles at the same instant, they contract simultaneously.

Disorders of the heart beat may result from disturbances in the S-A node or conduction system, or from some abnormal focus of excitation in the auricular or ventricular musculature These are schematically shown in Figure 21 1 For convenient reference they are discussed not solely on the basis of point of origin but according to the following classification

1 Tachycardia.
2. Bradycardia
3. Disturbances of conduction.
4. Arrhythmias.

There is some overlapping. In auricular fibrillation, for example, one finds arrhythmia and usually tachycardia. In heart block, there may be bradycardia, arrhythmia, or both.

Although heart beat disturbances can sometimes be correctly identified by history, physical examination, or fluoroscopy, it is always wise to confirm the diagnosis by electrocardiogram Often the latter is essential.

TACHYCARDIA

Sinoauricular Tachycardia

In adult males at rest, the normal range of heart beat is 60–80 per minute, in females 70–90, but figures outside these limits are not necessarily an indication of anything amiss What might be considered normal in one person might in

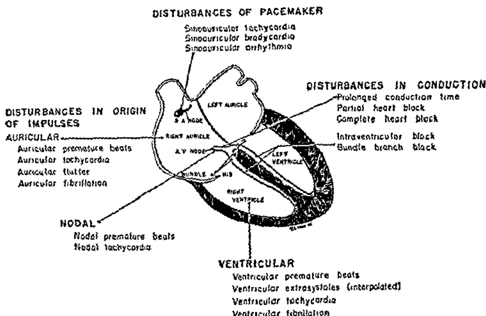


Fig 21 1 Diagram illustrating pathway of cardiac impulse and sources of common disturbances in rhythm

another be looked upon as abnormal When the rate is normally 60–70, an increase to 90 or 100 might be classed as tachycardia, but with a norm of 90, an increase to 100 would be regarded as insignificant

Sinoauricular tachycardia is simple, regular, accelerated heart action with a rate usually between 100 and 150, occasionally higher Onset and offset are gradual It occurs in response to exertion, excitement, pain, fever, or diminution of venous return, as in hemorrhage or shock, in thyrotoxicosis, following administration of certain drugs, and sometimes without demonstrable cause Under many of these circumstances it does not indicate heart disease However, tachycardia often occurs in heart disease or failure from any cause There are no symptoms except that the patient may be aware of the rapid rate. On examination one finds regular, rapid rhythm which may be further accelerated by exertion or diminished by some form of vagal stimulation.

Auricular Paroxysmal Tachycardia

Paroxysmal tachycardia may be auricular, A-V nodal, or ventricular in origin By far the most common, the auricular form, stems from an ectopic focus of increased irritability in the auricular muscle It is produced by a mechanism similar to that causing auricular premature beats, in fact, it may be considered as a series of rapid auricular premature beats It occurs in attacks which *always begin and end abruptly* They last from a few minutes to several days—usually a few hours—and may appear as often as several times daily or as infrequently as once every few years

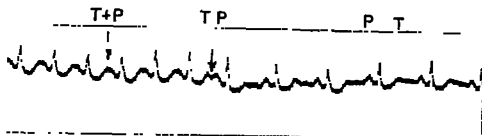


FIG 21 2 Auricular paroxysmal tachycardia Lead 2 Rate 160 Second arrow indicates point of reversion to normal sinus rhythm, rate 100 In complexes prior to reversion, each P wave is superimposed on preceding T wave Sagging of ST segments is due to recent digitalis administration

Symptoms

The nature and severity of symptoms vary, depending largely on the duration of the episode and status of heart muscle The patient suddenly becomes aware of palpitation and a rapid heart beat *which persists throughout the attack* He may complain of precordial ache and a feeling of tightness in the chest, exhaustion or faintness, and perhaps dyspnea. Apprehension is common At the sudden termination of the episode he is likely to experience one or two strong thumps within the thorax followed by disappearance of the palpitation and awareness of return of normal rhythm.

Signs

The heart rate is usually between 180 and 200 *Rhythm is absolutely regular,* once the abnormal rate is established it remains fixed throughout the attack. If the apical rate is repeatedly counted for a few minutes, it will be always exactly the same. Exercise, breath holding, change of position, or any other factor which alters the rate in S-A tachycardia has no influence during an episide One notable exception, however, is that an attack can at times be abruptly terminated by exertion of pressure over a carotid sinus or the eyeballs, drinking a glass of ice water, vomiting, or by some other procedure which stimulates the vagus nerve. During a long episode, the pulse may become thready and blood pressure low; if is is exceptionally long or the patient has underlying heart disease, congestive failure may develop.

Except where underlying heart disease exists, auricular paroxysmal tachycardia causes no permanent harm Once the attack is over, the patient is as well as ever, although he may feel exhausted for a while. In the presence of actual heart disease each episode imposes added strain on the myocardium, thus contributing to ultimate failure.

Differential Diagnosis

From the S-A type, auricular paroxysmal tachycardia is differentiated by the following characteristics

1. A more rapid rate than is usually found in S-A tachycardia.
2. Abruptness of onset and offset

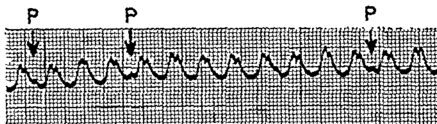


FIG 21 3 Ventricular paroxysmal tachycardia in a man age 45 with acute posterior myocardial infarction Lead AVF Rate 200 *Bizarre, wide ventricular complexes Slight upward deflections indicated* by arrows represent auricular activity with a dissociated rate, other auricular deflections are lost in the ventricular complexes

3 Constancy of rate during attack with failure of such a measure as holding the breath, exercising, or changing position to create even a minor change

4 Abrupt termination, in many cases, by vagal stimulation

The features which differentiate auricular paroxysmal tachycardia from other forms of tachycardia, auricular flutter, and auricular fibrillation will be indicated later.

Ventricular Paroxysmal Tachycardia

Resulting from a focus of increased irritability in the ventricular muscle, this may be considered comparable to a series of rapid ventricular premature beats Rate is somewhat slower (160–180) and rhythm slightly less regular than in auricular paroxysmal tachycardia The auricles beat independently, fibrillate, or are at a complete standstill. In contrast to the auricular form, ventricular paroxysmal tachycardia is a serious disturbance, usually occurring in association with severe cardiac impairment such as hypertensive heart disease or myocardial infarction In general its clinical features are similar to those of auricular paroxysmal tachycardia except that careful auscultation for several minutes may reveal, for a few beats at a time, slight irregularity of rhythm or variation in quality and intensity of the first sounds. Vagal nerve stimulation will never terminate an attack Electrocardiograms should always be taken to confirm diagnosis Because this form of tachycardia is usually superimposed on underlying heart disease, failure is likely unless the rate can be controlled

Auriculoventricular-Nodal Tachycardia

This is not common and usually not serious The impulses creating ventricular contraction start in the A-V node and usually by spreading in retrograde fashion cause corresponding auricular contractions Electrocardiogram is necessary for diagnosis

Auricular Flutter

Auricular flutter is most likely to occur in older persons, especially those with arteriosclerotic or hypertensive heart disease Once established, it is apt to last for months or years The auricles contract at a rate between 250 and 350, since

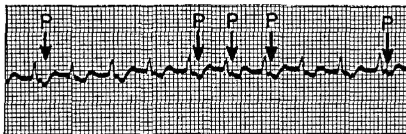


FIG 21 4 A-V nodal tachycardia Lead 2 Rate 160 P waves appear in the ST segments as a result of retrograde conduction

the ventricles cannot keep pace, functional A-V block results Usually the ratio of auricular to ventricular beats is 2 1 so that, with an auricular rate of 300, the ventricular rate will be 150 If the latter exceeds 175, the case is probably not one of flutter, for regular auricular contractions are rarely more than 350 The degree of block may vary from time to time so that fluctuations in ventricular rate are common. Furthermore the block may be sufficient to keep the ventricular rate within the normal range, in which event the disturbance may remain clinically unrecognized and be discovered only if an electrocardiogram chances to be taken.

Symptoms

If ventricular rate is rapid, the patient may be aware of it He may complain of precordial ache. Dyspnea and pain are largely dependent also on the status of the myocardium and coronary circulation; they are more likely with rapid ventricular rate Symptoms may remain latent if activity is moderately restricted but appear with increase of work load Added exertion may, by reducing the degree of block and raising ventricular rate, produce an episode of dizziness, faintness, or syncope, with or without congestive failure.

Signs

The most characteristic sign is the persistence for a long time of a rapid heart rate, usually in the neighborhood of 150 With the patient at rest it may change from time to time, is likely to be accelerated by exertion, and can be temporarily

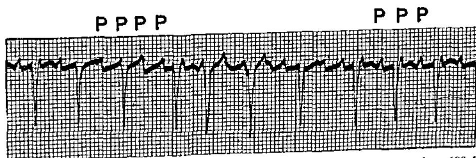


FIG 21.5 Auricular flutter Lead V1 Auricular rate 300 Ventricular rate varies from 100–150 due to changes in degree of A-V block

slowed by vagus stimulation, which has no effect on the auricular rate but slows the ventricles by increasing the block. If ventricular rate is normal, auricular flutter may be suspected if inspection of the cervical venous pulse shows it to be much more rapid than the ventricular rate. Indications of some underlying heart disease are usually found.

Differential Diagnosis

The following features of auricular flutter may be helpful in clinically distinguishing it from other forms of tachycardia.

Sinoauricular Tachycardia

1 Underlying heart disease is more likely to be present in flutter and there is not necessarily any factor such as acute infection or emotional strain to account for the rapid rate

2. If a change of rate can be created by vagal stimulation it will occur abruptly, whereas in S-A tachycardia it will be gradual

Paroxysmal Tachycardia

1 Flutter is likely to occur in older persons, especially those with basic heart disease

2 Attacks are of longer duration

3 The ventricular rate in flutter rarely exceeds 175, so that a rate higher than this points to paroxysmal tachycardia (A rate below 175 could be due to either)

4. Although vagal stimulation will temporarily slow the rate in flutter but never interrupt it, in paroxysmal tachycardia it will either stop an attack or have no appreciable effect

5 If, as is sometimes possible, the auricular rate can be counted by watching the neck veins, in flutter comparison with the ventricular rate will indicate a difference between the two

Auricular Fibrillation

Auricular fibrillation is common In a high percentage of cases it is found in association with underlying heart disease especially rheumatic or coronary disease, or untreated thyrotoxicosis. It is more likely in predominating mitral stenosis than other types of valvulitis and is uncommon in uncomplicated aortic valvulitis Paroxysmal or, rarely, permanent fibrillation may be encountered in persons with otherwise normal hearts; *here it is not regarded as serious*

The auricular musculature contracts irregularly and at a rate even higher than in flutter Due to the limitations of the conduction system the ventricles do not receive a regular flow of impulses; their contractions are *rapid* and *totally irregular* Without treatment, the ventricular rate is usually 120–150, occasionally slower. Except for the paroxysmal form encountered in otherwise normal hearts or when a correctible disturbance such as thyrotoxicosis is the cause, auricular fibrillation, once established, especially in chronic heart dis-

ease, is likely to be permanent. Reversion to normal rhythm may sometimes be accomplished by quinidine therapy.

Symptoms

The patient is usually conscious of abnormal heart action and may be dyspneic, especially during a paroxysmal attack or, in the permanent case, when the rate is rapid. If the rate is fundamentally slow or has been slowed by treatment, he is usually unaware of the trouble. Otherwise what symptoms are present are related more to the basic disease than to the fibrillation *per se.*

Signs

Irregularity of timing and strength of individual beats *is the distinguishing* characteristic.

Timing. Arrhythmia is complete, the intervals between successive beats never being the same. Sometimes contractions will follow each other in rapid succession, again, they will be separated by long pauses. Irregularity both of rhythm and force is more pronounced and consequently more easily discerned at higher than at lower rates.

Strength. Variations in force of contractions can be appreciated on auscultation as differences in intensity of sounds. Some ventricular systoles may be so weak that they fail to open the aortic and pulmonary valves and no second sound will be heard Others lack sufficient strength to produce pulsation in the peripheral arteries. This results in discrepancy between the heart rate determined by auscultation or palpation at the apex and that formed by palpation of the radial pulse (*pulse deficit*) In a pronounced case, the peripheral pulse may be only 70–90, auscultation at the apex will show the heart rate to be much higher, perhaps even 140–150 Lesser differences in strength of contractions will be detected when the blood pressure is taken (*see* Chap. 9). Because of this lack of uniformity, the systolic and diastolic blood pressure levels cannot be accurately determined, one uses the readings which apply to the greatest number of beats The record should show that they are approximate—and why.

When the ventricular rate is slow, slight differences in timing and force of beats may be detected by careful auscultation but can be overlooked Exercise might accentuate them sufficiently to clarify the picture; if not, the diagnosis would depend on the electrocardiogram.

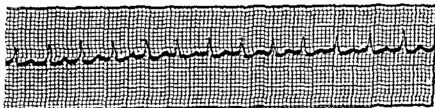


Fig. 21 6 Auricular fibrillation Lead 1 Ventricular rate approximately 200 P waves absent. *Because of rapid rate the irregularity of ventricular rhythm can be detected only by careful measurement.*

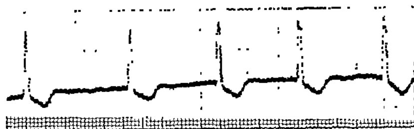


FIG [illegible] waves absent [illegible] of their descending [illegible]

Other features of auricular fibrillation which should be noted are.

1 *Digitalis therapy almost always slows the rate and reduces the irregularity of both strength and spacing of the ventricular contractions* If this response does not occur, one should be especially suspicious of active rheumatic infection or thyrotoxicosis

2. *Except in these two diseases, quinidine* therapy often, but not necessarily, restores normal rhythm. Since this response is by no means certain in any form of heart disease, the therapeutic test lacks the significance just described in connection with digitalis

3. *The character of murmurs in a case of* valvular heart disease may change with onset of auricular fibrillation. This is particularly true in mitral stenosis: due to absence of auricular systole the characteristic late accentuation of the diastolic murmur may be missing and sometimes the murmur is barely heard. With rapid ventricular rate the sounds heard may be so confusing as to make it impossible to determine the nature of the valvular defects. Only after therapy has slowed the ventricles will the sounds and murmurs be sufficiently clear to permit proper evaluation.

Differential Diagnosis

Auricular fibrillation is distinguished from other types of rapid rhythm by the total irregularity From marked sinus arrhythmia and frequent premature beats it may be differentiated by the response to exercise irregularity due to auricular fibrillation is likely to be increased, that due to others, decreased. Often one must rely on the electrocardiogram

BRADYCARDIA

SINOAURICULAR BRADYCARDIA

In a considerable number of well persons, heart rate is below average, perhaps as low as 40–50. Decreased activity of the S-A pacemaker, probably due to excess vagal stimulation, is responsible. There is no serious disturbance of the fundamental mechanism. This simple bradycardia is frequent in young adults, especially athletes in training, and occurs in some persons as a physiologic reaction during sleep. It is sometimes a familial trait. It may be temporarily in-

duced by vagal stimulation. It is common in myxedema, jaundice and mumps, occurs during convalescence from certain other infectious diseases, during the period immediately following labor, and with administration of certain drugs, particularly digitalis. Elevation of intracranial pressure is another cause; a downward trend indicates that the pressure is increasing and the patient's condition becoming more grave.

The differentiation between simple bradycardia and bradycardia due to heart block is discussed below.

DISTURBANCES OF CONDUCTION

Auriculoventricular Block

Impaired function of the A-V node or bundle of His hampers progress of the excitation wave to the ventricles Interference may be slight, merely slowing the impulse and thus prolonging conduction time between auricles and ventricles (*prolonged conduction time* or *first degree A-V block*); it may be sufficient to keep a certain number of impulses from reaching the ventricles at all (*partial* or *second degree A-V block*); it may be complete so that no impulses reach the ventricles (*complete* or *third degree A-V block*).

In *functional* block, the pathway is not diseased but because of its own refractory period is unable to convey impulses from the auricles to the ventricles as rapidly as it receives them This occurs in auricular flutter and auricular fibrillation In *structural* block, conduction fibers are damaged by some infections or degenerative process Digitalis and certain other drugs can also delay conduction

Prolonged Conduction Time. The interval between the onset of auricular and ventricular contractions is increased. This cannot be detected clinically The electrocardiogram shows prolongation of the P-R interval to more than 0.21 second, the upper limit of normal

During the course of active rheumatic infection, diphtheria or, less often, some other acute infectious disease, prolongation of conduction time may be the earliest and perhaps only indication of cardiac involvement Consequently, repeated electrocardiograms are essential. When it occurs during an acute infection delayed conduction usually disappears with recovery

Partial Heart Block. Impairment is sufficient to prevent a certain number of impulses from reaching the ventricles In lesser grades, as successive impulses traverse them, the conduction fibers become increasingly fatigued with gradual lengthening of conduction time until the point is reached at which an impulse from the auricles fails to reach the ventricles and a ventricular beat drops out (*Wenckebach's phenomenon*) This is indicated by a momentary interruption in ventricular rhythm During the pause, the bundle is rested and the cycle begins again. The initial A-V interval is near normal but as the bundle again becomes fatigued, progressive lengthening occurs until another beat is dropped. Depending on the degree of impairment, a beat may be dropped every sixth or seventh time or at longer or shorter intervals The block is designated by

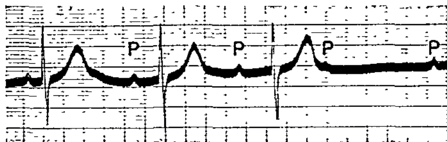


FIG 21 8 Wenckebach phenomenon Lead V2 Progressive prolongation of P-R interval At *termination of third T wave note upward deflection representing an auricular contraction which is* not followed by a ventricular response.

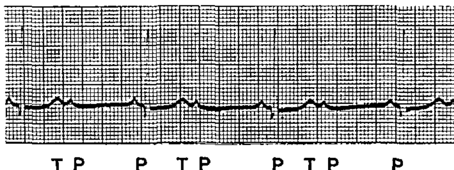


FIG 21 9 Partial heart block with 2:1 rhythm Lead 1 Auricular rate 94 Ventricular rate 47 Only every other P wave is followed by a ventricular complex

expressing the ratio of auricular to ventricular contractions: if every seventh beat is dropped one has 7 6 heart block, if every fourth beat, 4.3. With greater damage, one may find 3 2 or 2 1 block, 3 1 and 4 1 block are possible but rare

In all but the 2 1 and higher degrees of block, rhythm is irregular and clinical diagnosis usually not difficult. On auscultation one should detect an interruption of rhythm each time the ventricle misses. Since the beat is usually dropped at regular intervals, one finds a *regular irregularity* This *is one of the causes* of so called "intermittent pulse"

Irregularity of peripheral pulse due to heart block must always be differentiated from another, much more common, but less important cause of irregularity—premature beats which lack the strength to produce pulse waves at the periphery A weak premature beat will almost always be audible over the precordium but with dropped beat, no sound, or only the faintest sound of auricular contraction, will be heard

When the block is 2·1 or higher, the ventricular rate is *slow* and *regular.* If the auricular rate in 2 1 block is 70, the ventricular rate is 35, if the auricular rate is 80, the ventricular rate will be 40 It may be possible, by careful auscultation, to hear, between the ventricular beats, the faint sounds of the auricular contractions which occur without ventricular responses

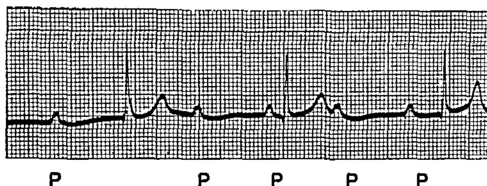


FIG 21 10 Complete heart block Lead 2 Auricular rate 95 Ventricular rate 40 Complete dissociation between P waves and QRS complexes A regularly due P wave is lost in the first QRS complex

In simple bradycardia, the ventricle responds to an accelerating stimulus by a *gradual* rise in rate, and, following withdrawal of the stimulus, by a *gradual* fall. In high-grade partial block such a stimulus usually reduces the block so that the response is a *sudden* and *striking* increase in rate with a *sudden* return to the previous figure after its withdrawal. Furthermore, abrupt changes sometimes occur spontaneously in partial block: a 2 1 rhythm, for example, may shift to 3:2, 7:6 or even to an occasional dropped beat In complete block, as noted below, an accelerating stimulus creates *little if any* change of rate.

Complete Heart Block. Although S-A impulses continue to activate auricular contractions, none of them reach the ventricles The auricles maintain their normal rhythm, usually 70–80 The ventricles contract in response to impulses presumably initiated in the A-V node or bundle of His. Rhythm is regular, rate usually between 28 and 40. It may be as low as 10 or as high as 50. Auricular and ventricular systoles are completely disassociated, occurring at varying moments with respect to each other

The diagnosis of complete heart block is usually not difficult. *No other disorder shows such a slow regular beat and such little change of rate and rhythm by measures which ordinarily accelerate the ventricles.*

By listening closely, one may detect changes in heart sounds from beat to beat based on the varying time relationships between contractions of the auricles and ventricles When auricular systole occurs approximately midway between two ventricular systoles its faint sound may be heard. When it falls immediately before a ventricular systole, the first sound may be reduplicated; when it occurs simultaneously with a ventricular systole the first sound will be somewhat intensified. Jugular pulsation due to auricular contraction can sometimes be observed during the long pause between ventricular beats Detection of these minor variations is extremely difficult, it is wiser to rely on the electrocardiogram.

Ordinarily, heart block per se produces few symptoms. The patient may be conscious of dropped beats or of sudden change in rhythm created by shift in degree of block. Whether or not other indications of cardiac impairment will be present depends on the nature and degree of the underlying trouble.

In complete block, the ventricular beat may become temporarily slower—even as low as 10–20. A moderate drop will create light-headedness, dizziness, or, possibly, momentary unconsciousness; a pronounced drop or one of long duration may cause syncope and convulsions (*Adams-Stokes syndrome*). The latter is not common.

The importance of heart block is that it usually indicates serious myocardial damage. It may be the only indication of coronary disease, or of cardiac involvement in rheumatic infection, diphtheria, or, rarely, some other infectious disease, including syphilis.

Bundle Branch Block

The excitation wave is delayed or blocked in the left or right branch of the bundle of His so that the impulse fails to reach the two ventricles simultaneously (*left* or *right bundle branch block*) As a result, the QRS complex is widened and distinctive QRS complex and T wave changes are created (*see below*) It is generally accepted that prolongation of the QRS interval to 0.11 second constitutes *incomplete* bundle branch block; to 0 12 second or more, *complete* bundle branch block. The term *intraventricular* block is applied to cases in which the QRS interval is prolonged to any degree but the typical pattern of left or right bundle branch block as described below is not present.

Bundle branch or intraventricular block may be encountered in coronary, hypertensive, valvular and congenital heart disease, as well as in various forms of infectious and toxic myocarditis Neither necessarily indicates serious trouble and by itself cannot be used as a prognostic criterion In fact, such a conduction defect is sometimes found without demonstrable cause in persons showing no other evidence of heart disease.

Since there is no variation of rate or rhythm, the block cannot be recognized clinically, although a diastolic gallop or reduplication of the first or second sound is suggestive In routine study diagnosis is made only by electrocardiography At times, the changes created by bundle branch or intraventricular block will obscure those caused by myocardial infarction and interfere with the electrocardiographic diagnosis of this disturbance

Left Bundle Branch Block.

Left axis deviation, usually.

QRS complexes 0 11 sec. (incomplete); 0 12 sec. or more (complete)

Leads 1 and AVL R waves notched and slurred.

Leads V1 and V2 R waves small or absent; S waves wide and deep

Leads V5 and V6. R waves wide and notched; S waves absent.

All leads: T waves of asymmetrical form and usually opposite in direction to main QRS deflection (as determined by width—not height). ST segments usually depressed where T waves are inverted.

Right Bundle Branch Block.

Right axis deviation, usually.

QRS complexes: 0 11 sec (incomplete); 0 12 sec. or more (complete)

Leads 1 and AVL: S waves wide and slurred but usually not deep

Fig 21 11 Left bundle branch block QRS 0 12 sec Note characteristic changes in ventricular complexes as described in text

Leads V1 and V2· R waves large and notched or RSR′ complexes present
Leads V5 and V6. S waves wide and slurred
All leads. T waves and ST segments show same variants as in left bundle branch block.

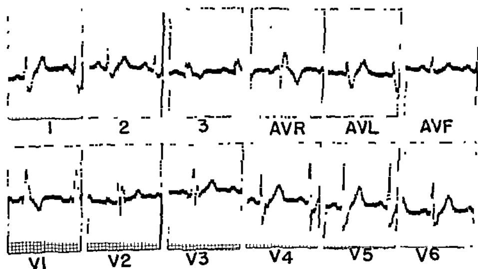


Fig 21 12 Right bundle branch block QRS 0 12 sec Note characteristic changes in ventricular complexes as described in text

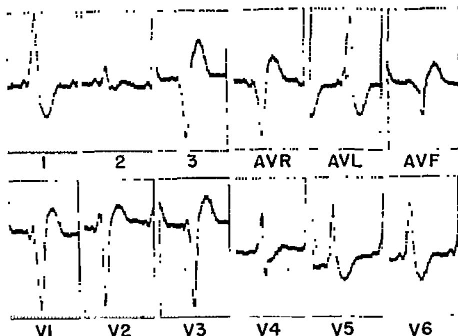


FIG 21 13 Wolff-Parkinson-White syndrome Short P R intervals (0 08 sec) Prolonged QRS intervals (0 14 sec) QRS and T patterns resemble those seen in left bundle branch block

Differentiation of left from right bundle branch block is best made in leads V1, V2, V5 and V6

Intraventricular Block.

QRS complexes 0 11 sec or more, with slurring and notching

T waves Non-specific changes in one or more leads may or may not be present

Wolff-Parkinson-White Syndrome

Detectable only by electrocardiography, this syndrome is not clinically recognizable P-R intervals are short, 0 1 second or less, QRS complexes wide, and, along with T waves, similar in appearance to those encountered in bundle branch block These variations in timing offset each other, for the interval between onset of P and termination of QRS is normal. Spontaneous reversion to normal P-R intervals, QRS complexes and T waves may occur periodically; the time between onset of P and termination of QRS remains unchanged. Although the mechanism is not understood, most authorities believe that the excitation wave either travels more rapidly than normally from the auricles to one ventricle or the other by way of some special conducting fibers, or that it spreads with abnormal rapidity down one of the bundle branches. Persons with this syndrome are especially subject to various forms of paroxysmal tachycardia; serious trouble is uncommon

ARRHYTHMIA

Sinoauricular Arrhythmia

Heart rate gradually increases with each inspiration and gradually decreases with each expiration, probably as a result of vagal influence on the S-A node. The fluctuation is accentuated by deep breathing, diminished by accelerated heart action Sinus arrhythmia is found in most children and many normal adults It is of no clinical importance except as a source of unnecessary concern to a mother who takes her child's pulse or the adult who takes his own. When it is unusually pronounced or, as sometimes happens, rhythm changes occur abruptly, it may be confused with auricular fibrillation. In sinus arrhythmia acceleration of heart rate will diminish the irregularity; in auricular fibrillation, increase it.

Rarely, an extreme type of sinus arrhythmia is encountered in which inhibition of the node is sufficient to cause the heart to pause for an abnormally long period (*cardiac standstill*). The symptoms are similar to those of the Adams-Stokes syndrome. Sudden death from cardiac standstill may occur in heart disease, especially coronary artery disease, in syncope from any severe vagotonic reaction irrespective of the presence of heart disease, and without heart disease, during anesthesia, an operation, or peripheral circulatory failure, or following trauma, suffocation, or electric shock Rarely it may follow administration of quinidine or a toxic dose of digitalis

Premature Beats

Ectopic beats arise from some abnormal focus in the auricles, ventricles or A-V node and are designated accordingly. They are the most common cause of disordered rhythm but are relatively unimportant and do *not* indicate serious heart disease. They are often called extra systoles, but the term is misleading, as they are rarely "extra." They may occur singly, at rare or frequent intervals, sometimes as often as every other beat (*coupling* or *bigeminal pulse*) or every third beat (*trigeminal pulse*). Short runs of successive premature beats may entirely replace normal contractions, a variant closely allied to paroxysmal tachycardia

Often no cause is found. They are just as apt to occur in persons without heart disease as in those who have it Sometimes they are induced by coffee, tea, tobacco, alcohol or digitalis. During digitalis administration, appearance of coupled rhythm due to ventricular premature beats is a definite indication of overdosage.

A patient with premature beats usually complains of palpitation, "skipping" or "turning over" of his heart The sensation may be felt precordially, substernally, or in the throat Some describe a feeling that the heart is "about to stop," an impression attributable to the compensatory pause described below For obvious reasons, it is essential in taking a history, to distinguish these sensations from the substernal oppression, tightness or pain of angina pectoris.

Premature beats can usually be identified on auscultation. the regular rhythm

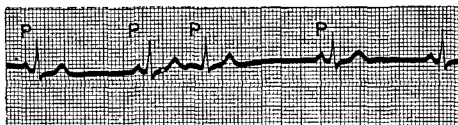


FIG 21 14 Auricular premature beat Lead 2 Third P wave is premature and differs in configuration from the others It is followed by a normal ventricular complex

is interrupted from time to time by a beat which occurs earlier than expected and may or may not be followed by a compensatory pause A run is marked by a series of quick beats appreciably faster than those of the basic rhythm If ectopic beats occur frequently or in runs, the aberrant rhythm may be mistaken for auricular fibrillation With exercise, irregularity due to ectopic beats will be reduced because the normal pacemaker will initiate impulses at a faster rate, thus nullifying the influence of the abnormal focus, whereas in auricular fibrillation, irregularity will be increased. Since some ectopic beats are faint one must exclude them by careful auscultation before making a diagnosis of dropped beats. Because of the relative harmlessness of premature beats and the serious import of heart block and auricular fibrillation, an electrocardiogram must always be taken in an equivocal case

Auricular Premature Beats. In response to an abnormal focus of excitation in an auricle, auricular systole occurs earlier than would be the case if the muscle were being activated by a normal impulse originating in the S-A node Once *started, the wave follows the* usual pathway, thus creating an early ventricular contraction The normal impulse, which meanwhile has started in the S-A node, is nullified but the next one creates a normal contraction which follows the premature beat after a time interval similar to that between two normal beats Since the interval between a regular beat and a following premature beat is shorter than normal and that between *the premature beat and the* following regular beat is normal, it is obvious that their sum is less than the sum of two regular intervals Dominant rhythm is thus altered at this point, a variant which does not occur with ventricular premature beats, as will be indicated below

The electrocardiogram will show abnormal P wave followed by a slight prolongation or, rarely, shortening of the P-R interval and normal or slightly altered ventricular complex.

Ventricular Premature Beats. If an abnormal focus of excitation is in a ventricle, the ventricles contract before the normal excitation wave from the auricles can reach them When the latter does arrive the ventricles are unable *to respond because they are already contracting* or are in the refractory state following their premature activity. This normal impulse is consequently wasted, the ventricles must wait for the next regular impulse to arrive from the auricles

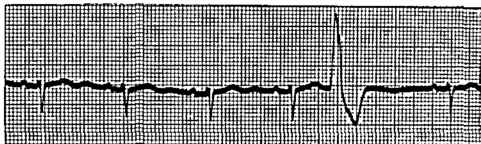


FIG 21 15 Single ventricular premature beat Lead AVF Fifth ventricular complex early, of bizarre pattern, and followed by a compensatory pause Regularly-due P wave is lost in the premature ventricular complex

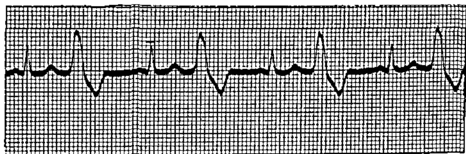


FIG 21 16 Ventricular premature beats creating bigeminy Lead AVL Normal complexes with their preceding P waves are readily distinguished from the wide bizarre complexes originating in the ventricle This rhythm is often found with excessive dosage of digitalis

before contracting again. Since auricular rhythm remains undisturbed, a single ventricular premature beat is followed by a pause longer than that existing between two normal beats (*compensatory pause*). But the interval between a regular beat and a succeeding premature beat added to the compensatory pause will equal two normal intervals In other words, the interval between one beat and the second subsequent beat will be the same irrespective of whether the intervening ventricular systole is normal or premature. Recognition of the compensatory pause will serve to distinguish a ventricular from an auricular premature beat, except in the rare instance when the latter is followed by a pause slightly longer than the intervals of the basic rhythm

Rarely, a ventricular premature contraction will appear in the interval between two normal successive beats (*interpolated ventricular beat*). This can correctly be called an *extra systole*. Obviously there is no compensatory pause since the extra contraction is squeezed into a single diastolic interval

On the electrocardiogram the ventricular complex of the premature beat will be very different from normal QRS deflection, perhaps in the normal direction, perhaps reversed, will be of longer duration than normal. The following T wave will be abnormally large and opposite in direction to the main QRS deflection. The P wave representing the auricular contraction at this point is usually lost in the abnormal ventricular complex. The interval between the

normal beat preceding and the normal beat following the premature contraction will be equal to two regular intervals.

Nodal Premature Beats. These are rare. The aberrant impulse arises in the A-V node and may pass upward and downward initiating simultaneous contractions of auricles and ventricles. The next regular beat follows the premature beat after the normal interval so that there is no compensatory pause. The electrocardiogram will show a normally conducted but premature ventricular complex with a distorted P wave appearing shortly before, within, or just after the QRS complex.

22

MISCELLANEOUS CARDIOVASCULAR DISORDERS

THYROID DISEASE

Thyrotoxicosis

Increased cardiac activity and vasodilatation are almost always present Symptoms related to the heart, depending on the degree and duration of disease, range from simple palpitation or awareness of rapid rate to severe dyspnea and other indications of congestive failure. In the typical thyrotoxic case the following cardiovascular manifestations are to be expected

Tachycardia. A rate of 100–120 or more even with the patient at rest is the rule; it will not diminish during sleep, as in tachycardia due to other causes, including neurocirculatory asthenia and emotional stress Continuous or paroxysmal *auricular fibrillation* occurs in about 30 per cent of cases and, when discovered in a patient without other evidence of heart disease, should always suggest thyrotoxicosis

Forceful Heart Action. This is indicated by a snapping and diffuse apex impulse, a palpable sensation resembling thrill over the precordium, and a loud snapping first sound

Murmurs. An apical systolic murmur probably due to slight left ventricular dilatation may be present In addition, one may hear under the manubrium a harsh systolic sound resembling pericardial or pleural friction, it is regarded as due to rubbing together of normal pericardial or pleural surfaces resulting from overactivity of the heart and associated dilatation of the pulmonary artery

Blood Pressure Changes. As a rule, systolic pressure is somewhat elevated, diastolic diminished, and pulse pressure increased accordingly

Peripheral Vascular Dilatation. This is manifested by moist, warm skin and, in severe cases, by such peripheral vascular signs as Corrigan pulse, capillary pulse, pistol shot sound in the femoral artery, and a diastolic murmur heard distal to the point of pressure, if pressure is applied over a large artery.

Cardiac Enlargement. As a rule, the heart is enlarged only when congestive failure has developed, but vigorous heart action may, when one percusses, give him the mistaken impression that the heart is enlarged before this change has occurred.



FIG 22 1 Myxedema Rate 50 Low voltage of P waves and QRS complexes T waves low or inverted (A comparable pattern but with normal or increased rate may be found in nutritional deficiency)

The fluoroscopist will observe rapid heart beat with perhaps increased amplitude of pulsations and prominence of hilar shadows. Enlargement is not significant except in advanced cases The electrocardiogram will show rapid rate *but no specific changes*

In the presence of the characteristic manifestations of thyrotoxicosis, such as loss of weight, exophthalmos, tremor, restlessness, overactivity, and sweating, *the diagnosis of thyrotoxic heart disease presents no difficulties* If these indications are not striking, the clinical picture may be more suggestive of some cardiac disturbance Predominance of palpitation, dyspnea, snapping first sound, murmurs, and tachycardia with or without auricular fibrillation, or congestive failure might lead to an erroneous diagnosis, especially of mitral stenosis. Thyrotoxicosis must always be considered in a patient with tachycardia, paroxysmal or permanent auricular fibrillation, or congestive failure, especially if response to digitalis therapy is not obtained. In a doubtful case, basal metabolism and serum protein-bound iodine and radioactive iodine studies should be pursued.

MYXEDEMA

Cardiac rate is slow and sounds less distinct than normal, conveying the impression of sluggish heart action Enlargement may be evident. Impaired function or even congestive failure may develop in the severe case Typically, x-ray shows some enlargement, slow rate and pulsations of diminished amplitude *Electrocardiographically*, the well-established case will show sinus bradycardia, low voltage of QRS complexes and flattening of P and T waves Return to normal can be effected by therapy.

ANEMIA

In severe anemia, angina pectoris, cardiac dilatation, lack of cardiac reserve, or even congestive failure may develop. Often the enlargement and murmurs are

sufficiently pronounced to cause a mistaken diagnosis of structural heart disease.

In the presence of profound anemia, cardiac signs and symptoms must not be attributed to heart disease unless they persist after treatment has restored the blood to normal.

TRAUMA

Penetrating Wound

A stab wound of the heart or perforation by a fractured rib or other cause usually results in death from hemorrhage and cardiac tamponade due to hemopericardium Occasionally healing will take place but subsequent rupture of the weakened area of myocardium is possible

Chest Injury

A direct blow on the chest or a crushing injury may, in rare instances, cause immediate rupture of the normal heart even though the thoracic cage itself is not badly damaged In other cases death will occur days to months after injury as a result of heart rupture, or failure secondary to myocardial contusion or rupture of a valve leaflet, a papillary muscle, or the interventricular septum In still others, impairment of cardiac function due presumably to myocardial or coronary artery injury seems to persist for weeks or months, the patient's activity is restricted by dyspnea or angina pectoris. Some patients recover, others develop increasing incapacity Symptoms can be due to neurocirculatory asthenia or cardiac neurosis precipitated by the trauma; to distinguish between one of these and actual cardiac injury is extremely difficult unless one can demonstrate evidence of structural change by physical examination or electrocardiography. The most likely electrocardiographic variant is alteration of T waves.

Although trauma of this kind is not common, it is assuming constantly increasing importance because of the frequency of automobile crashes (*steering wheel injury*)

In the presence of pre-existing heart disease of any type, a blow, crush or sudden strain may increase cardiac impairment or even precipitate failure

Arteriovenous Fistula

A direct communication is established between an artery and its accompanying vein, usually by a penetrating wound, less often by rupture of a traumatic or congenital aneurysm. Unless the opening is small, shunting of blood from arterial to venous side imposes strain on the left ventricle, leading to functional impairment, enlargement, and eventual congestive failure. The vein becomes dilated with consequent valvular incompetence and may show arterial pulsation; return flow distal to the fistula is inhibited If the lesion is in an extremity, the part distal to it may show weak or absent arterial pulse, coldness, and other evidence of impaired blood supply. The most important sign is a loud continu-

ous murmur with systolic accentuation best heard over the fistula but transmitted to a considerable distance chiefly proximally. A thrill may be felt. Increased pulse pressure due to lowered diastolic, increased systolic pressure or both, is possible Angiocardiography is often helpful in demonstrating the lesion.

NUTRITIONAL DISORDERS

Dietary Deficiency

In beriberi, pellagra, and other disturbances such as chronic alcoholism, peptic ulcer, persistent vomiting and chronic diarrhea in which food intake or absorption is limited, cardiovascular changes are likely Lack of thiamin is the accepted cause

The clinical picture is variable Onset may be gradual or sudden. One may find simple tachycardia, bradycardia or, in severe cases, evidence of impaired function or even failure of either or both ventricles The most serious cases may also show evidence of peripheral circulatory failure. Except in the early stage, x-ray will show a generally dilated heart with small, rapid pulsations, a shadow which does not conform to the more usual patterns of heart disease, and pulmonary congestion out of proportion to x-ray evidence of heart disease. Although the electrocardiogram may be normal, some change is the rule, most likely flattening or inversion of T waves, prolongation of Q-T interval, and low voltage In all but the most severe cases, the cardiovascular complications are reversible, normal circulation can be restored by adequate doses of thiamin

Whenever cardiac insufficiency cannot be attributed to one of the usual causes, dietary deficiency must be considered, especially in the presence of other manifestations of avitaminosis such as polyneuropathy, pellagrous oral or cutaneous lesions, anemia, gastro-intestinal disturbances, or evidence of inadequate food intake or utilization.

Electrolyte Imbalance

Hypernatremia. Increased sodium in the body may, by causing fluid retention, result in peripheral and pulmonary edema, and congestive failure. This complication is most likely in heart, renal, or liver disease, especially if treatment with parenteral fluids is injudiciously regulated, but can occur without any of these diseases if fluid and electrolyte balance is not properly maintained Sodium retention may also result from treatment with a ketosteroid, androgenic or estrogenic hormone Increased blood volume due to overdosage with blood or a blood substitute may cause circulatory embarrassment or failure even though the cardiovascular system is normal and electrolytes are not out of balance

Hyponatremia. Decreased sodium from diminished intake or excessive loss, as in profuse sweating, diarrhea, prolonged vomiting or gastric drainage, or overtreatment with a mercurial diuretic will cause lowered blood pressure and perhaps tachycardia Skeletal muscular weakness and often cramps, gas-



Fig 22 2 Nutritional deficiency due to chronic alcoholism in a man age 35

A Film taken while patient showed clinical picture of congestive failure Heart enlarged Diffuse hazy density of lung fields due to edema

B After 2 weeks of treatment with large doses of vitamins and other appropriate measures Heart shadow smaller Lung fields have cleared. Complete recovery after 8 weeks Ten-year follow-up Total abstinence and no indications of any cardiovascular disorder

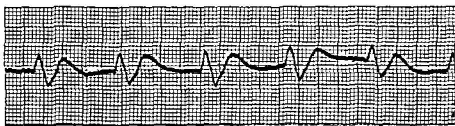


FIG 22.3 Hyperkalemia Lead I Bradycardia P waves absent R waves low and QRS complexes widened

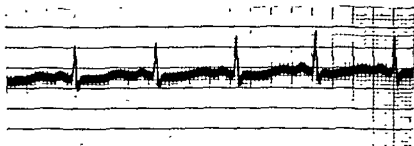


FIG 22 4 Hypokalemia due to intractable diarrhea Lead AVF Q-T intervals markedly prolonged (0 56 sec) T waves low and broad Q-T prolongation largely due to widening of T waves

tro-intestinal disturbances, and mental confusion are common Nitrogen retention may accompany the syndrome Edema occurs in the so-called salt-losing nephritic but here it is due to lowered serum protein In adrenal insufficiency, lowered sodium level due to excessive excretion results in generalized weakness, dehydration, gastro-intestinal disturbance, small heart with feeble action, perhaps tachycardia, and low blood pressure Rarely, the gastro-intestinal symptoms may predominate to such an extent that a diagnosis of an acute intra-abdominal disease such as appendicitis may be incorrectly entertained Congestive failure is rare, peripheral failure common

Hyperkalemia. Increased potassium usually results from anuria or overtreatment with potassium, especially if renal function is impaired The characteristic electrocardiographic changes are lowering or absence of P waves, peaking and increased amplitude of T waves, sinus bradycardia, or perhaps ventricular arrhythmia usually due to ventricular ectopic beats Auricular standstill and prolonged, slurred QRS complexes may appear in advanced cases. Clinically, one may note characteristic severe muscular weakness and, if present, ventricular premature beats. Death from cardiac standstill is the principal hazard

Hypokalemia. Diminished potassium is most often seen following prolonged vomiting, gastric drainage, or diarrhea, especially if potassium replacement is not initiated. It may occur after rehydration in diabetic coma or during protracted steroid therapy. The electrocardiogram often provides the earliest indication. Characteristic changes are prolongation of the Q-T interval with

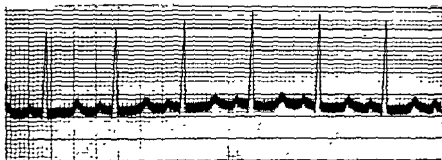


Fig 22 5 Hypocalcemia Lead AVF Prolongation (considering heart rate) of Q-T intervals (0 40 sec) largely due to lengthening of ST segments T waves normal

lowering or inversion of T waves The Q-T prolongation is to a large extent a reflection of broadening of the T wave In severe cases depression of S-T segments and prominent U waves may also be observed Death from hypokalemia is usually due to respiratory failure or peripheral vascular collapse rather than to cardiac failure

Hypocalcemia. Found in severe renal disease and hypoparathyroidism, diminished calcium content of the blood is reflected in the electrocardiogram as prolongation of the ST segment with resulting widened Q-T intervals

Obesity

Because of his dyspnea and perhaps swelling of his lower extremities, the obese person is often mistakenly regarded as having heart disease Actually his heart is handicapped by infiltration of fat between its muscle bundles, the added work load due to excess body weight, and enlargement of the peripheral vascular bed resulting from increase of adipose tissue and body surface With weight reduction, the dyspnea and edema will disappear.

TOXIC AGENTS

Digitalis

The physiologic effect of this drug is reflected in the electrocardiogram as prolongation of P-R interval, sagging of ST segments, and lowering or in-

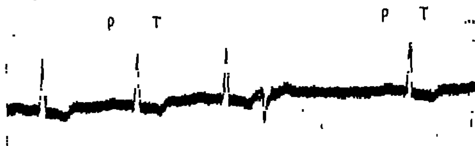


Fig 22 6 Digitalis intoxication Lead 1 P-R intervals prolonged (0 26 sec). ST segments depressed T waves inverted. One ventricular premature beat present.

version of T waves. Ventricular premature beats may appear. In excessive dosage the changes become more pronounced; one may find partial or complete heart block, frequent ventricular premature beats, often with bigeminal rhythm, or perhaps ventricular tachycardia. Rarely, sudden death from standstill or ventricular fibrillation will occur.

Quinidine

Although valuable in the treatment of auricular fibrillation and certain other types of disturbed rhythm, quinidine may produce undesirable effects, such as ventricular arrhythmia or some form of heart block. It *occasionally* causes sudden death, presumably from depression of the S-A node, ventricular fibrillation or respiratory paralysis. The characteristic electrocardiographic findings are broadening of P waves, prolongation of P-R interval, and broadening of QRS and T complexes with prolongation of the Q-T interval

Tobacco, tea, coffee or alcohol may, in some persons, induce premature contractions, S-A or paroxysmal tachycardia, or paroxysmal auricular fibrillation Drugs other than those mentioned may adversely affect the heart, but their discussion is not within the scope of this book

NEOPLASM

Primary tumor of the heart is rare; when present it is most likely a benign myxoma of a valve. Metastatic tumor, although not quite so rare, is unimportant clinically because it is not often recognized and is overshadowed by the disease elsewhere in the body

Direct or lymphatic invasion of the pericardium from an intrathoracic tumor such as bronchiogenic carcinoma or some malignant disease of the mediastinal lymphnodes is occasionally encountered If the pericordium becomes sufficiently infiltrated, thickened and fixed, the picture may be comparable to constrictive pericarditis (*see* Chap 23)

IDIOPATHIC CARDIAC FAILURE

We use this term to designate a small but important group of cases characterized by advancing and eventually fatal heart failure from unknown cause Occurring at any age between infancy and mid-life, but mostly in young adults, the picture is one of progressive dilatation and hypertrophy, increasing functional impairment, and death from congestive failure, usually within 1 to 3 years from onset of symptoms *Post-mortem* examinations show varying degrees of hypertrophy and dilatation without demonstrable cause. In some cases enlargement is the sole finding Others show diffuse fibro-elastic thickening of the endocardium, especially of the left ventricle, but without valvular involvement. In infants, this variant is regarded as congenital (*congenital endocardial fibro-elastosis*), in other age groups, whether it is congenital or

acquired is not known. Occasional cases present diffuse subacute or chronic inflammatory changes in the myocardium without evidence of inflammation in the other layers of the heart or elsewhere (*isolated or Fiedler's myocarditis*)

NEUROCIRCULATORY ASTHENIA (EFFORT SYNDROME)

The responses of the normal person to unaccustomed exertion are breathlessness, palpitation, sometimes substernal ache, weakness and exhaustion. Neurocirculatory asthenia is a disturbance in which less than the normal amount of effort provokes these symptoms, yet *no structural disease of the heart is present to account for them.* They are common in normal persons following acute infectious disease, operation or some emotional shock. Here, they disappear with time

The syndrome is also frequently encountered in a chronic form and without any precipitating factor, especially in highstrung persons. Often one can elicit a history of overprotection during childhood and youth, inadequate performance, and perhaps poor adjustment to environment. The patient may have been guarded for years because of "heart trouble," the incorrect diagnosis of which may well have been based on the detection of nothing more than an insignificant murmur He has led a sedentary life—an adjustment to limited exertional tolerance, and fear. Any added physical or emotional load precipitates symptoms or increases those already present.

Although persons in this category are often seen in civilian practice, they are particularly likely to be encountered in military hospitals In general, those with emotional instability and the poorest performance records in civilian life are most likely to break during the training period or while serving behind the lines. But the syndrome also develops acutely in troops during combat, even those with no history of previous trouble, here it is a response to prolonged physical fatigue and battle strain.

Symptoms

The usual complaints referable to the cardiovascular system are

Palpitation. The patient is conscious of rapid and forceful, rather than irregular, heart action.

Precordial Pain. Apical or precordial ache, or recurrent sharp, stabbing pain at the apex is a common complaint The patient does not describe substernal discomfort, oppression, smothering, or other sensation characteristic of angina pectoris.

Dyspnea. This is more subjective than objective and usually is associated with frequent sighing. The latter is an important factor in the diagnosis of neurocirculatory asthenia.

Other common symptoms are weakness, fatigue, fainting attacks, nervousness, flushing, sweating, and disturbances referable to other systems such as vague indigestion, headache and backache.

Signs

Although one or more of the following signs may be observed, there is no definite evidence of actual heart disease.

Forceful Cardiac Action.

Tachycardia. In contrast to the tachycardia of thyrotoxicosis, the rate usually diminishes during sleep.

Precordial Tenderness.

Suggestion of Apical Systolic Thrill. Actually not a true thrill, this is due to diffuseness of the apex impulse

Soft Systolic Apical Murmur.

Labile Blood Pressure.

Indications of nervous instability such as tenseness, tremor, quick response, clammy hands and feet, and sweating are usually found

A patient with actual heart disease will sometimes present symptoms of cardiac insufficiency which are out of proportion to the degree of actual heart damage, they are the result of neurocirculatory asthenia, precipitated by the patient's worry over the disclosure that he does have heart disease

Since so many of the manifestations of neurocirculatory asthenia are equally characteristic of heart disease and of thyrotoxicosis, differential diagnosis must be made with extreme care, especially because their respective therapeutic requirements are so different

DISSECTING ANEURYSM OF AORTA

Partial rupture of the wall of the aorta, usually a short distance above the aortic ring or less often near the site of the ligamentous arteriosum, occurs from an intimal tear overlying an area of degeneration in the media. Rarely, atheromatous or other lesion of the intima is at fault The primary trouble is in the media Syphilis is not a factor. Blood penetrates partway through the media, finds a plane of cleavage and may dissect backward into the pericardium or along the pulmonary vessels, forward along the course of the aorta, or both Blood flow into any of the branches of the aorta, including the subclavian, renal and common iliac arteries, may be impeded by direct pressure or by extension of the dissection within their walls As a result, manifestations of the disease are protean.

The episode almost always occurs beyond the age of 40, is more frequent in males, and rarely seen in the absence of long-standing hypertension Agonizing pain, beginning abruptly with or without preceding physical or emotional stress, and variously described as rending, tearing or crushing, is the outstanding symptom It usually begins in the front, sides or back of the thorax, extends rapidly to involve all of the thoracic cage, perhaps the neck and jaws, and often travels downward into the abdomen and legs The arms may be spared It lasts for hours or days and is not entirely relieved by large doses of morphine or other potent analgesics Onset is accompanied by peripheral

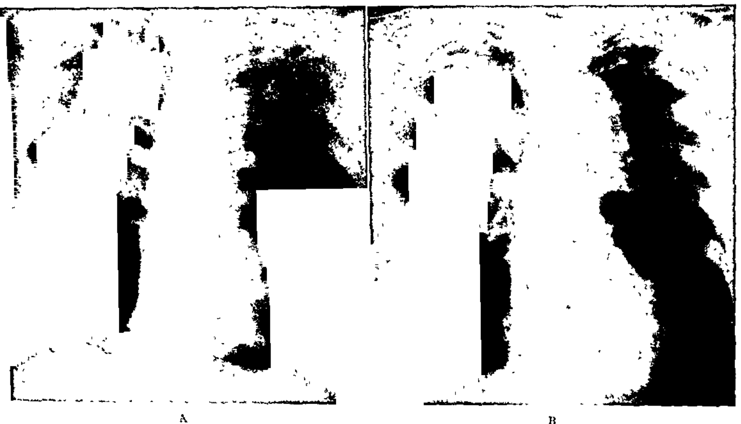


Fig 22 7 Dissecting aneurysm

A Routine film in a man age 60 with hypertension Heart and great vessels essentially normal

B Six years later Pronounced widening of aorta beginning at arch and extending distally almost to level of diaphragm Postmortem examination 1 year after this film was taken showed a channel of dissection from level of left subclavian artery to a point just above celiac axis where it reentered the lumen This channel was endothelialized

circulatory failure with severe prostration, pallor, sweating, and tachycardia, but blood pressure is likely to remain elevated. Slight to moderate fever and leukocytosis are the rule. One usually finds evidence of impaired circulation to other parts, such as loss of consciousness, sensory or motor disturbances, absence of arterial pulsations in one or more extremities, abdominal distention, or anuria. The patient usually dies within a few hours or days, sometimes after a longer interval, with signs of hemorrhage and shock, due to rupture of the aneurysm into the pericardial sac, a pleural cavity, or the abdomen. In rare instances, blood may break back into the lumen of the aorta lower down; the patient will survive for months or years, eventually dying of a second rupture or some other disease.

X ray shows widening of an unusually long segment of the aortic shadow and perhaps deposits of calcium situated within the shadow at some distance from its outer edges.

Dissecting aneurysm is very likely to be confused with acute myocardial infarction. It may be possible to distinguish it from this much more common insult by, (a) abruptness, severity and wide distribution of the pain, (b) persistence of hypertension after rupture has occurred, (c) evidence of impaired circulation to other parts of the body, and (d) absence of the progressive electrocardiographic changes which occur in myocardial infarction.

PERICARDITIS

Pericarditis occurs in the following forms: (1) Acute: (a) fibrinous, and (b) pericarditis with effusion. (2) Chronic: (a) simple fibrous, (b) fibrous with extensive adhesions, and (c) constrictive

ACUTE PERICARDITIS

Fibrinous Pericarditis

This is encountered as a complication of various infectious diseases, most likely rheumatic fever, tuberculosis, pneumonia, or sepsis It may occur in any of the so-called collagen diseases, especially lupus erythematosus and rheumatoid arthritis, and in some, if detected, may be an important substantiating diagnostic feature It is common, for reasons not understood, as a terminal complication of chronic nephritis with uremia (*see* Chap 32) Localized pericarditis due to myocardial infarction (*see* Chap 20) and acute benign pericarditis presumably due to virus infection (*see* Chap 18) have already been discussed.

Symptoms

Fibrinous pericarditis may produce no symptoms or cause mild or severe pain over the precordium, in the left shoulder or epigastrium The pain may be increased by inspiration Because it lessens discomfort, the patient may tend to remain fixed in a particular position, most likely sitting upright, bent forward or to one side, or lying on one side.

Signs

Pericardial Friction Sound. This is pathognomonic One hears a rough, grating or *shuffling sound which occurs irregularly during each part of the* cardiac cycle, overlapping rather than exactly coinciding with either systole or diastole (*see* Chap. 12).

Evidence of Diaphragmatic Irritation. Breathing is likely to be shallow, grunting, or irregular. Upper abdominal pain, tenderness, and muscle tenseness may be severe enough to suggest some acute intra-abdominal disturbance

Electrocardiographic Findings

The characteristic changes are slight ST elevations with diphasic or inverted T waves in most leads. Often they are difficult to distinguish from those of

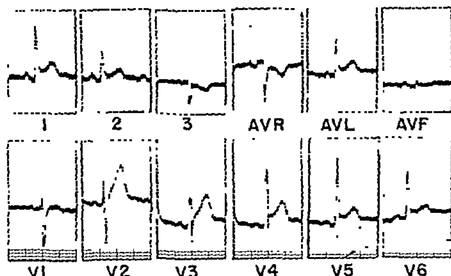


FIG 23.1 Acute pericarditis Elevation of ST segments in Leads 1, 2, AVL and V2-V6

coronary insufficiency Probably a reflection of disease in the subpericardial zone of the myocardium, they last from days to months.

Diagnostic Pitfalls

Acute fibrinous pericarditis may be overlooked because of failure to detect friction sound, or the diagnosis is incorrectly made by confusing pericardial friction with one of the sounds indicated below. In the seriously ill patient, auscultation is likely to be hampered by ronchi, grunting, or noisy breathing from other cause The character of the pain and other manifestations may be such as to make the disease distinguishable with difficulty from myocardial infarction, an acute pleuropulmonary disturbance, or some upper intra-abdominal insult such as ruptured peptic ulcer or acute pancreatitis

Pleural Friction. Fibrinous exudate covering that part of the pleura which overlaps the heart may give rise to sounds closely resembling those of pericardial friction Heart movement causes the inflamed pleural surfaces to grate against each other This is sometimes called pleuropericardial friction, but the term should be avoided because the sounds arise in the pericardium or the pleura, not both. Pleural sounds are often louder during deep inspiration, diminished toward the end of expiration. Pericardial sounds, on the other hand, are usually best heard when the breath is held at the end of expiration. *If friction sounds heard in the precordial region cease altogether when the breath is held, they are not dependent upon friction between visceral and parietal pericardium and must be of pleural origin.* Sometimes the distinction cannot be made with certainty.

Cardiac Murmurs. Pericardial friction sounds can usually be distinguished from physiologic or pathologic murmurs by the fact that they do not exactly correspond either with systole or diastole, do not constantly occupy any one

portion of either, and are scratchy. Murmurs are more regular, seem less superficial, vary less from day to day and with changes in position, and are not increased by pressing the stethoscope against the chest wall.

Friction Sounds Unassociated with Inflammation. When faint friction sounds are heard over the heart, especially along the left border of the sternum, one must bear in mind that they may be due, not to inflammation of the pericardium, but to dehydration or to abnormal pressure exerted on the pericardial surfaces They are especially likely to be audible as a result of *forceful heart action and dilatation of the pulmonary artery in thyrotoxicosis (see* Chap 22) and, rarely, in acute cor pulmonale due to massive pulmonary embolism.

Pericarditis with Effusion

If fibrinous pericarditis other than that associated with nephritis or myocardial infarction fails to heal promptly, serous, purulent, or bloody fluid is likely to accumulate in the sac The quantity may vary from just a little to more or many times that normally present

Serous Effusion Serous *effusion of appreciable degree may occur* in *any* of the disturbances causing fibrinous pericarditis except nephritis and myocardial infarction. It is most likely due to rheumatic infection, acute benign pericarditis, or tuberculosis

Purulent Effusion. Purulent effusion appears as a complication of some pyogenic infection, especially lobar *pneumonia or septicemia,* and is serious because of toxic absorption as well as mechanical hindrance

Hemorrhagic Effusion This bespeaks malignant disease or, less often, tuberculosis. It must be distinguished from hemopericardium.

Hemopericardium Hemopericardium can be caused by puncture wound, rupture of the heart, retrograde dissection in dissecting aneurysm, or hemorrhagic pericarditis in a patient receiving anticoagulant therapy. Bleeding may be so rapidly fatal that no physical signs become recognizable

Hydropericardium Hydropericardium, in which the fluid is a transudate, is encountered in association with generalized edema in congestive failure, renal disease, and other maladies showing marked fluid retention

The manifestations of pericardial effusion, as indicated below, vary with the amount of fluid, the rapidity of its *development, and its location. If fluid* develops *slowly,* 1000 cc or more may be contained in the cavity without serious impairment of cardiac action, and hence with no significant symptoms or signs of circulatory embarrassment On the other hand, if accumulation is *rapid* and the pericardium has no time to distend for accommodation of the *fluid, a much smaller amount will cause* inflow stasis and restricted diastolic expansion of the heart *(cardiac tamponade)* This happens most frequently in hemopericardium, rheumatic infection, and septic pericarditis. Furthermore, a considerable quantity developing posteriorly may not *create the alterations*

of percussion borders of apex impulse which are to be expected if the fluid also appears anteriorly Paracentesis is often necessary for diagnosis.

Symptoms

Pain is usually less severe than during the fibrinous stage. Dyspnea and other indications of cardiac embarrassment may be present. Pressure on an adjacent structure may cause dysphagia, aphonia or irritating cough.

Signs

Whenever fibrinous pericarditis is present, one should be on the alert for the development of effusion Disappearance of friction sound may indicate separation of the two pericardial layers by fluid, it does not necessarily mean improvement Depending on its location and speed of development, a large quantity should cause some but not necessarily all of the signs indicated below. Detection of a small effusion, 300 cc or less, is not possible unless it develops quickly enough to cause tamponade Nor is recognition important unless hemorrhagic or purulent fluid is regarded as likely.

Alterations of Heart Borders. These changes do not occur if the fluid is confined posteriorly Otherwise, one may find·

1. Extension of percussion dulness for a considerable distance to the left beyond the point of maximal cardiac impulse

2. Extension of dulness further to the right than occurs with cardiac enlargement except with extreme dilatation of the right auricle Displacement of the heart to the right must be excluded Although dulness in the fifth intercostal space just to right of the sternum, producing an obtuse cardiohepatic angle, is often said to be an important sign, we do not so regard it.

3 Extension of left upper border Normally dulness extends only to the third intercostal space; in pericardial effusion it may reach to the second or higher

4. Modification of area of dulness by positional changes Turning the patient from one side to the other or changing him from upright to supine is supposed to create shifts in the borders of dulness based on flow of fluid to the most dependent part of the pericardial cavity Actually, we believe these procedures are of little value because free movement of fluid is so often hampered by its own thickness or by intrapericardial adhesions Even by x-ray such changes are rarely striking enough to be regarded as significant

Variation in Apex Implise. If fluid collects anteriorly between the heart and chest wall, the impulse will become diminished or perhaps disappear. If most of it is posterior, the change may not occur

Diminution of Heart Sounds. The sounds will be poorly heard, provided there is an appreciable amount of fluid anteriorly.

Indication of Pressure on Adjacent Part. A small area of dulness, bronchial breathing, and increased tactile fremitus near the angle of the left scapula due to compression of lung by fluid (*Ewart's sign*) is common These findings

sometimes lead to a mistaken diagnosis of pneumonia. Dyspnea, dysphagia, aphonia or irritating cough may result from pressure on the pertinent structure

Signs of Tamponade.

1. Increase in venous pressure indicated by distention of cervical veins and perhaps enlargement of the liver. In a doubtful case, venous pressure should be determined by the direct method (*see* Chap. 8)

2 Pulsus paradoxus, diminution of volume, or disappearance of peripheral pulse during inspiration (*see* Chap 9) If peripheral circulatory failure and labored respiration can be excluded, well-established pulsus paradoxus almost certainly points to cardiac compression from pericardial fluid or constriction.

3. Diminution of systolic blood pressure with consequent lowering of pulse pressure.

4 Feebleness or absence of cardiac pulsation on fluoroscopy.

X-ray Findings

The heart shadow is enlarged, globular, and shows loss of characteristic configuration The right cardiophrenic angle remains sharp. In contrast to congestive failure, the hilar shadows are not prominent On fluoroscopy, cardiac pulsations are diminished or absent. Rarely, a film taken with the patient supine may show minor differences when compared to one taken with him upright· the supracardiac shadow will be somewhat wider and the right border not so far to the right.

Electrocardiographic Findings

The picture is similar to that found in acute fibrinous pericarditis

Diagnostic Pitfalls

Cardiac Enlargement. In hypertrophy and dilatation, the left border of dulness does not extend as far beyond the apex impulse, nor the right border as far laterally as in pericardial effusion Diminution of apex impulse or heart sounds favors fluid. X-rays are important When the two disturbances coexist, demonstration of fluid is particularly difficult. If daily observations show rapid widening of the cardiac area, effusion is likely, but the cause might be acute dilatation, as in severe acute rheumatic infection or failure from other cause. Signs of cardiac tamponade point to effusion.

Localized Pleural Effusion. Occasionally encapsulated or interlobular empyema or pleural effusion may be mistaken for pericardial effusion. The exudate is confined to an area in the left axilla just beyond the apex impulse and produces signs of compression of the left lung similar to those of pericardial effusion Thorough study, including careful x-ray examination, is necessary Usually, a patient with pleuritic effusion is less toxic than one with pericardial effusion and shows fewer signs of cardiac embarrassment. If both pericardial and pleural fluid are present, it is impossible to detect the former until the latter has been withdrawn.

Lobar Pneumonia. If a patient with fever, leukocytosis and other signs of



FIG 23 2 Pericardial effusion Heart shadow enlarged and globular Right cardiophrenic angle sharp

infection is found to have signs of pulmonary consolidation near the tip of the left scapula, a mistaken diagnosis of lobar pneumonia may be made; actually the pulmonary signs are due to compression of lung by pericardial fluid

CHRONIC PERICARDITIS

SIMPLE FIBROUS PERICARDITIS

This is one of the end-results of acute fibrinous or serofibrinous pericarditis The antecedent inflammation, usually rheumatic, occasionally tuberculous or other, may have been so mild as to have caused neither signs nor symptoms Since it causes no circulatory embarrassment, fibrous pericarditis usually exists unrecognized and is found incidentally, *post mortem*. There may be nothing more than areas of thickening on the cardiac surface (*milk spots*) or light fibrous adhesions between the two pericardial surfaces Sometimes firm adhesions totaly obliterate the cavity These are harmless unless complicated by constriction of the heart or strong external adhesions to other parts

FIBROUS PERICARDITIS WITH EXTENSIVE ADHESIONS

Usually of rheumatic origin, this is marked not only by obliteration of the cavity but also by pronounced thickening of the pericardium and strong fibrous attachments to adjacent chest wall, diaphragm or mediastinal structures (*mediastinopericarditis*) It is highly probable that, in rheumatic infection, involvement of the pericardium does not occur independently, but only as one of the features of pancarditis *Post-mortem* studies support this view, extensive fibrous pericarditis is rarely found without valvulitis The need created by

adhesions for the heart, during systole, to move the diaphragm, chest wall or mediastinal structures, is an important, but rarely if ever, the sole factor in cardiac embarrassment, enlargement, and eventual failure. Valvular changes and perhaps myocardial damage are also operative.

Symptoms

Depending on the degree of cardiac embarrassment, these range from complete absence to the indications of congestive failure.

Signs

When found, any of the following signs suggests this form of pericarditis.

Systolic Retraction of Chest Wall.

1 In region of apex impulse Retraction may be more noticable during *inspiration. Its value as a sign of adhesive pericarditis is limited by the fact* that it may be observed in cases of markedly hypertrophied or overactive heart, especially in thin persons.

2. Below angle of left scapula (*Broadbent's sign*) Both the ribs and intercostal spaces may be affected. This too is sometimes caused by a greatly enlarged heart without adhesions

3 *At tip of sternum. If the heart is fixed in this area, it will tend to rise* rather than descend during inspiration and, consequently, to draw the xiphoid process inward

Fixation of Heart. The 2 to 5-cm shift of the apex impulse which occurs in the normal person when he turns from the right to the left lateral position or *vice versa* is prevented by the adhesions This may be determined by in*spection or palpation but often is apparent only by x-ray.*

X-Ray Findings

The heart shadow is usually enlarged and its margins are irregular and poorly defined No characteristic chamber enlargment is evident The shadow will not shift with change in the patient's position. On fluoroscopy, pulsations appear diminished in amplitude and perhaps absent in some parts of the shadow The movements of the heart with respiration are abnormal. Normally, during expiration, it seems to become wider and its apex to flatten out on the diaphragmatic surface, during inspiration it becomes narrower and the apex seems more pointed With fixation, these changes do not occur Nor does the heart as seen in the lateral view change position with respect to the sternum as it normally does during inspiration. instead of appearing further away from the sternum it seems to follow it upward and forward. Especially in the left oblique or left lateral views, calcium deposits may be seen, most likely on the diaphragmatic surface or in the sulcus between auricles and ventricles Presence of calcium is a pathognomonic sign of marked fibrous pericarditis.

Electrocardiographic Findings

Low voltage, especially of QRS complexes and lowering, flattening, or slight inversion of T waves in all or most leads are usually present. The picture may

be confused by the indications of hypertrophy or other changes secondary to the associated valvular defects.

Chronic fibrous pericarditis, even when adhesions are extensive, is often overlooked during life unless fixation of the heart or pericardial calcinosis is demonstrated. The circulatory changes are apt to be attributed solely to the accompanying valvular disease. In the unlikely event that extensive adhesive pericarditis exists without valvulitis, it can just as easily be overlooked; the murmurs of relative valvular change secondary to the enlargement, can be mistakenly attributed to structural valvular disease

Constrictive Pericarditis

The two pericardial layers are firmly adherent, greatly thickened, and contracted Many of these cases are of tuberculous origin and usually occur without evidence of the disease elsewhere. The etiology of the remainder is unknown. Rheumatic infection is rarely, if ever, a cause—an important point in distinguishing constrictive pericarditis from the adhesive type just described

Restricted diastolic expansion of the ventricles due to the encasement results in diminished diastolic inflow The latter, in turn, leads to elevated venous pressure and stasis, both peripheral and pulmonary (*inflow stasis*) Impaired filling obviously causes diminished stroke output Limited systolic force, due to restriction of heart action by the rigid pericardium or to some degree of myocarditis, may also contribute to diminished output.

Symptoms

Dyspnea on effort is the commonest initial and predominating symptom; in contrast to other forms of heart disease, orthopnea is not striking. Other complaints which may appear at onset or later are swelling of the legs and abdomen, easy fatigability, palpitation, persistent cough, and upper abdominal discomfort due to liver engorgement or ascites.

Signs

Feeble Apex Impulse. This is a reflection of impaired cardiac action

Absence of Pronounced Enlargement. *The heart is normal sized or even* small, rarely, slightly enlarged Determination of heart size by physical examination is difficult because the apex impulse is feeble or absent and percussion borders may be obscured by coexistent pleural thickening or fluid One must rely on x-ray.

Absence of Murmurs. Significant murmurs are not the rule.

Indications of Inflow Stasis. Venous stasis is manifested by engorgement of the veins of the neck and upper extremities, enlargement of liver, ascites, dependent edema, and often uni- or bilateral hydrothorax Restricted diastolic filling of the left ventricle with consequent pulmonary hypertension causes accentuation of pulmonic second sound and sometimes rales in the lungs, and other indications of pulmonary engorgement. In contrast to congestive failure from most causes, the peripheral signs are out of proportion to the indications

of trouble in the lesser circulation; liver enlargement and ascites are out of proportion to the peripheral edema. Cyanosis may be present but is not a predominant sign.

Pulsus Paradoxus.

Low Blood and Pulse Pressures.



FIG 23 3 Constrictive pericarditis Arrows point to increased density reflecting calcific changes in pericardium Right costophrenic angle obliterated by chronic pleuritis and pleural effusion

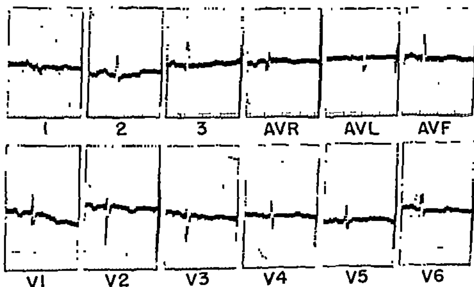


FIG 23 4 Constrictive pericarditis Low voltage. T waves flattened or inverted in most leads.

X-Ray Findings

The heart shadow is normal, small, or slightly enlarged. *Calcification* can often be seen in the pericardium, most likely anteriorly over the lower portion of the heart. Hilar vessels are usually engorged By fluoroscopy, diminished amplitude of cardiac pulsation is observed and calcinosis not evident on the film may be detected

Electrocardiographic Findings

Low voltage, especially of QRS complexes and lowering, flattening, or slight inversion of T waves are the rule

Diagnostic Pitfalls

Congestive Failure. This is rarely found without appreciable cardiac enlargement and significant murmurs, and, in most cases, striking, pulmonary engorgement.

Liver Failure. Enlargement of the liver, ascites and dependent edema occurring without the usual signs of heart disease may lead to an erroneous diagnosis of cirrhosis of the liver or perhaps acute hepatitis Distention of the veins of the neck and upper extremities, physical and x-ray evidence of *restricted* cardiac action, pericardial calcinosis, and electrocardiographic changes point to constrictive pericarditis.

24

DISEASES OF THE TRACHEA AND BRONCHI

THE TRACHEA

Acute Tracheitis

Acute infection of the respiratory tract may be limited to the nasopharynx, spread to the throat, tonsils and accessory sinuses, or progress further downward, causing acute laryngitis, tracheitis, bronchitis, or even bronchopneumonia. In any of these, constitutional symptoms—fever, malaise, headache, bone or muscle aches—can be mild or severe

Acute tracheitis refers to infection which extends to the trachea but presumably no further Diagnosis depends on the occurrence of cough and a feeling beneath the sternum of discomfort, rawness or oppression, any of which is aggravated by inspiration of cold air Because of associated laryngitis the cough is likely to be raspy. There are no characteristic physical signs.

Chronic Tracheitis

In chronic tracheitis, usually associated with chronic bronchitis, the symptoms and signs are those of bronchitis Some observers describe a form of chronic tracheitis which may exist independently as a result of constant inhalation of smoky or soot-laden air. It is characterized by mild cough and redness, edema, and thin or thick tenacious secretion of the mucous membrane, seen through the bronchoscope

Tracheal Obstruction

The trachea may be partially or totally occluded by·

1 Foreign body.

2 Pressure on its wall, as from tumor of lung or mediastinum, or enlarged thyroid gland

3 Disease of its wall such as tumor, tuberculosis, or *syphilis*

Gradual or partial obstruction is marked by harassing cough, progressive dyspnea and eventually transient spasms of near-suffocation caused by temporarily increased obstruction secondary to edema and retained secretion Breathing is often stridulous and becomes very labored in the late stages. Dyspnea is both inspiratory and expiratory More difficulty may be experienced in ex-

pelling than inspiring air with resultant abnormal pulmonary inflation, indicated by hyperresonance and diminished, normal, or emphysematous breath sounds. Diffuse thickening of the tracheal wall, a mass in the airway, or deformity due to an extrinsic mass may be demonstrable by x-ray

Complete obstruction is fatal unless it can be promptly relieved by intubation or tracheotomy. Inhalation of foreign body is the most common cause of sudden, complete obstruction

BRONCHITIS

Acute Bronchitis

Symptoms

When infection starting in the upper air passages reaches the larger bronchi, the patient is likely to complain of substernal pain similar to that of tracheitis but extending somewhat lower and felt in the area corresponding to the anatomic position of the primary bronchi Cough is more severe and may occur in paroxysms Constitutional symptoms are, as a rule, more pronounced than in acute tracheitis Cough and substernal pain may herald acute bronchitis, developing as a complication during the course of measles, typhoid fever, malaria, and other infectious diseases

If the smaller bronchi become involved, cough and substernal discomfort are more distressing, constitutional reaction greater and moderate dyspnea, often with wheezing, may develop Sometimes breathing becomes definitely asthmatic and respiratory distress severe

Signs

Rales. By the coarseness or fineness of the rales can be estimated the caliber of the bronchi affected Low-pitched, groaning, or loud, bubbling rales (*rhonchi*) indicate involvement of the larger tubes, squeaking, whistling, or fine or medium moist rales, the smaller tubes When the process is limited to the large bronchi, one will find no signs, or rhonchi which disappear after cough. Early involvement of the smaller tubes is indicated by diffusely distributed sibilant and sonorous rales which vary from time to time both in character and distribution and are frequently changed by cough Later, as secretion becomes less viscid, they are replaced by crepitant rales

Diminished Breath Sounds. The breath sounds are vesicular but may be poorly heard because of interference with flow of air into the lungs or masking by rhonchi and rales.

Acute bronchitis is usually bilateral Occasionally, in influenza, acute sinusitis, or other acute respiratory infection, fine moist rales appear over an area of lung perhaps the size of the palm of the hand, they may persist for weeks but eventually disappear Such an area is often called localized bronchitis but probably represents a focus of atypical or bronchopneumonia, and not pure bronchitis Either diagnosis must be made with caution, for *persistent rales in*

a limited area are much more apt to mean pulmonary tuberculosis or bronchiectasis One should never be satisfied with a diagnosis of acute bronchitis or pneumonia if the rales persist in a limited area for more than a few days or at most, a few weeks

Diagnostic Pitfalls

Pulmonary Tuberculosis. *In the usual type of chronic ulcerative pulmonary* tuberculosis, the rales are for the most part confined to one or more localized *areas, almost* always the apices, and are persistent. In acute bronchitis they are diffusely distributed and disappear in a short time. In miliary tuberculosis, as in acute bronchitis, the rales are widely distributed, but in the former constitutional symptoms are much more severe X-ray, examination of sputum and gastric contents, and course of the disease should establish diagnosis.

Bronchial Asthma. Here, as in the early stage of bronchitis, rales are sibilant and sonorous but respiratory distress is greater and evidence of infection lacking.

Cardiac Asthma. This is paroxysmal, signs of heart disease are present

Edema of Lungs. As in the later stages of bronchitis, rales and diminished vesicular breath sounds may be heard but they are usually confined to the dependent portions, that is, posteriorly if the patient has been lying on his back, or at the lung bases if he has been sitting up for some time Diagnosis is facilitated if one finds evidence of myocardial weakness or some other cause for the edema

Bronchopneumonia. The physical signs of diffuse bronchopneumonia may be chiefly those of the coexisting bronchitis and its presence may be impossible *to detect by physical signs* X-ray *may or may not be helpful, depending on the* size of the bronchopneumonic patches. Bronchopneumonia should be suspected *in every patient with the physical manifestations of bronchitis but with a more* severe systemic reaction than usual

The term *capillary bronchitis* has been used *to describe a disturbance more* serious than ordinary acute bronchitis and marked by pronounced constitutional reaction, high fever, leukocytosis, distressing cough, dyspnea, cyanosis, and fine moist rales throughout the lungs It seems likely that areas of alveolar involvement too small to be detected by physical or x-ray *examination are present* and that these cases should be regarded as bronchopneumonia.

Chronic Bronchitis

Chronic bronchitis is usually a secondary manifestation *of some more serious* illness It may be primary in persons constantly exposed to dust, such as stonecutters or coal miners, or result from repeated attacks *of acute bronchitis over* a long period It is also seen in association with chronic circulatory insufficiency. Many cases of emphysema or intractable asthma also *ultimately develop chronic* bronchitis, presumably as a result of complicating bacterial infection. Where there is chronic bronchitis there is often some *degree of emphysema and* bronchiectasis, and *vice versa*. The three are often found together, recurrent

attacks of superimposed acute bronchitis and bronchopneumonia, especially during cold weather, are frequent Any case of bronchitis should be investigated for other associated or underlying disturbances.

Symptoms

The chief symptom is cough, with or without sputum It is much worse in winter and sometimes almost disappears during the summer. The sputum varies from slight amounts of mucoid material to large quantities of mucopus; the latter probably reflects associated bronchiectasis. Several factors influence it, among them being duration of the disease, season, and amount of bronchiectasis

Dyspnea of some degree is usually present; there may be wheezing

Signs

Generalized sibilant and sonorous rales are the rule In addition, one may hear medium or fine moist rales, especially at the bases. Both types are subject to seasonal variation. Indications of associated emphysema or bronchiectasis may also be found Except in the presence of some complication, x-ray is not helpful

Chronic bronchitis is easy to recognize Its especial importance is that it directs attention to the possible presence of some more important ailment which usually underlies it

Bronchial Asthma

The term *asthma* has been loosely applied to attacks of respiratory distress—especially of the paroxysmal type—no matter what the cause. Actually, *asthma*, or preferably *bronchial asthma*, refers only to a particular disturbance, the chief manifestation of which is severe paroxysmal dyspnea of the expiratory type and which is presumably due to hypersensitivity to some extrinsic or intrinsic substance, usually a protein In the study of a case of suspected asthma the history is particularly important, especially as it pertains to description of the attacks, hereditary factors, other allergic phenomena such as hay fever or urticaria, and seasons of the year and environmental circumstances when attacks predominate

*The precise mechanism by which the dyspnea is produced is not fully under*stood It is probably related to either or both of the following.

1 Diffuse narrowing of the finer bronchi by spasm.

2 Edema of the mucous membranes of the finer bronchi and secretion of mucoid exudate into their lumens.

The frequency and severity of attacks are contingent chiefly on the sensitiveness of the patient, the degree of exposure, and the frequency of contact with the offending substance Episodes occur in some cases as often as several times daily, in others, as infrequently as once or twice in a lifetime, although such a *rarity is uncommon* Some patients are never entirely free of trouble, experiencing acute exacerbations on top of a background of persistent respiratory difficulty of varying degree

An acute attack may last from a few minutes to as long as several days Some cases are of such severity that spasm and progressive accumulation of exudate cause alarming impairment of aeration and may result in death Intractable chronic asthma often becomes complicated by emphysema, chronic bronchitis, and perhaps bronchiectasis. Happily, these do not ordinarily develop in patients with recurrent attacks of acute asthma unless the episodes occur with great frequency.

Symptoms

Onset of the typical acute attack is sudden, offset gradual Symptoms are almost always worse at night

Dyspnea. This is the outstanding symptom. It is accompanied by a feeling of suffocation and constriction in the chest. It is expiratory; outflow of air is chiefly impaired so that the lungs remain distended.

Cough. During the early stage this is harassing and productive of only a very small amount of sputum which is grayish-white, semitransparent, tenacious, and contains small pellets of opaque substance (*Laennec's pearls*) On microscopic examination these are found to be spiral filaments of mucus containing cellular elements (*Curschmann's spirals*) The most important microscopic finding is the presence of eosinophiles in large numbers Later, cough becomes looser and productive of profuse and watery sputum unless secondary infection has occurred, in which event the sputum will be mucopurulent.

Signs

Labored Respiration. The rate is increased, respiratory movement obviously forced and accompanied by wheezing which is usually audible at a distance Movement of the thorax is limited. Accessory muscles become active

Hyperresonance. This is accompanied by reduction of cardiac and hepatic dulness and low position of the diaphragm

Diminished Fremitus.

Diminished Breath Sounds. Although of normal quality, the inspiratory phase is short, the expiratory phase prolonged Sometimes the sounds are barely heard because of associated rales.

Rales. Sibilant and sonorous rales are heard throughout the thorax Toward the end of an attack, fine crepitant rales sometimes appear at the bases

Cyanosis. This will be apparent if pulmonary ventilation is sufficiently impaired

X-Ray Findings

The lung fields are large and emphysematous The diaphragm is low, flattened, and shows limited mobility, especially during expiration Areas of pneumonitis, atelectasis, or localized air trapping may be observed

Diagnostic Pitfalls

Acute Bronchitis. Here one finds a history of upper respiratory infection and usually constitutional symptoms.

Cardiac Asthma. Since this is due to pulmonary hypertension, one will usually find mitral stenosis or some cause of left ventricular failure such as hypertension, coronary disease or aortic valvulitis.

Upper Respiratory Tract Obstruction. Dyspnea due to obstruction in a major air passage is both inspiratory and expiratory and usually accompanied by stridor Rales are not prominent

Partial Obstruction of a Bronchus. Here there are *unilateral* wheeze and *localized* obstructive emphysema, which differ from *generalized* wheeze and pulmonary distention seen in asthma (*see below*)

Bronchiectasis

This is fusiform, cylindrical, or saccular dilatation of the smaller, sometimes larger, bronchi The process may be limited to all or part of one or two lobes or be so widespread as to involve almost all of both lungs It probably never occurs as a primary disease although sometimes one can elicit no history of antecedent trouble to which it can be attributed It may follow pertussis, influenza, pneumonia, other respiratory infection, or appear as a complication of bronchial obstruction from any cause. Initially bronchiectasis is usually confined to a limited area but with time becomes more extensive by spread of infected material into previously healthy branches of the bronchial tree or by complicating attacks of acute bronchitis or bronchopneumonia Generalized bronchiectasis is frequently found in long-standing cases of chronic bronchitis or emphysema, local or general, in tuberculosis, and in other forms of chronic bronchopulmonary infection.

Just how the disease is produced is not clearly understood It is thought to be brought about by some degree of bronchial obstruction with resultant stagnation caused by infection of the bronchial walls Shrinking of surrounding lung parenchyma due to fibrosis may also be a factor. Whatever the mechanism, the end-result, in addition to dilatation, is thickening of bronchial walls, loss of elasticity, and retention of secretion The dilatations vary in size In advanced cases some reach a diameter of 1–2 cm. and communications between adjacent cavities may develop Severe grades are complicated by pulmonary changes, chiefly fibrosis, contraction, and perhaps chronic pleural thickening

Patients with bronchiectasis are particularly susceptible to repeated attacks of acute respiratory infection, bronchitis, or bronchopneumonia, especially during cold weather

Symptoms

Due to wide variation in degree and extent of the bronchial and pulmonary changes, there is a wide range of symptoms and signs. In the mildest cases they are almost negligible, in the severe ones they are pronounced Usually one will expect to find

Cough. Characteristically this occurs in paroxysms lasting from a few minutes to as much as an hour or two. Attacks are particularly apt to occur when the patient arises in the morning because of filling of the cavities overnight, and when he lies down or bends over because of the tendency of the cavities to drain

with change of position. Due to the associated bronchitis, cough is usually worse in winter.

Expectoration. In all but the mildest cases sputum is profuse and easily raised. The quantity varies; sometimes there is as much as 500 cc within 24 hours If the patient lies prone across the bed with both head and upper trunk lowered toward the floor, sputum can be raised with a minimum of cough or effort (*postural drainage*). Like *the cough, the amount of sputum is subject to* seasonal variation.

Grossly, the sputum, when fresh, is composed of discrete greenish-yellow or yellowish masses of mucopurulent material 1–2 cm. in diameter If allowed to stand for some time, the pus sinks to the bottom, and a layer of thin mucoid material remains above It is sometimes bloody In cases with marked stasis, the sputum may have a stale, musty odor but is not as foul as that of lung abscess or gangrene A loopful can be easily transferred to a cover slip—in contrast to the *more tenacious sputum of abscess Microscopically there are large numbers of* leukocytes and, often, ciliated cells Elastic fibers are rarely present Many varieties of bacteria abound intra- and extracellularly.

Hemoptysis. Ordinarily sputum is not bloody However, a single or recurrent attacks of frank pulmonary hemorrhage sometimes constitute the only symptom of the disease (*dry bronchiectasis*).

Dyspnea. This is dependent on the degree of associated bronchopulmonary disease

Pain. *This occurs only when there is an associated acute pleuritis or when* pleural adhesions are torn during a severe paroxysm of coughing.

Constitutional Reaction. This varies with the degree of retention and concomitant infection

Signs

Pulmonary Findings. These are contingent on the severity of associated pleuropulmonary disease They may change from time to time, depending on whether the diseased area is full of secretion or has recently been drained by cough. Rales may be the only local sign. They are moist, medium, or fine, if they are consonating, cavitation is suggested The percussion note is likely to be dull unless pulmonary or pleural disease is minimal Fremitus, breath, and voice sounds may reflect pulmonary infiltration or fibrosis, pleural thickening or emphysema Unaffected parts may show evidence of compensatory emphysema. Signs of trouble are sometimes obscured by overlying normal or diseased parenchyma

Retraction of Thoracic Wall. When a large area is diseased, pleuropulmonary contraction from fibrosis may pull in the thoracic wall and restrict respiratory motion locally Extensive change will cause displacement of heart and mediastinal structures, and gross thoracic deformity.

Clubbing of Digits. In a rapidly progressing acute case or a chronic case of long duration, clubbing of fingers and toes is common. Sometimes other indications of pulmonary osteoarthropathy appear

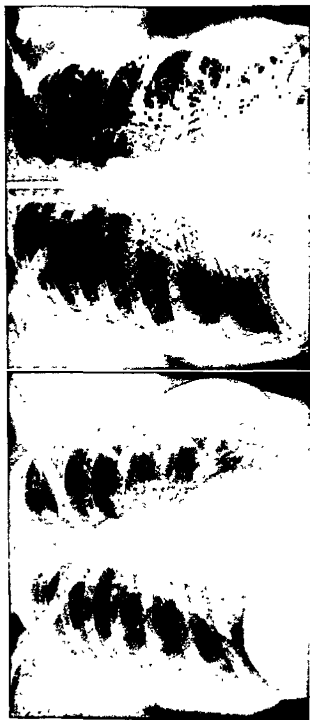


FIG. 24.1. Bronchiectasis in a girl age 17 with chronic productive cough beginning after attack of pertussis at age 4
A Plain film Normal appearance except for slight clouding in right lower lung field medially
B Bronchogram Normal bronchi outlined in left upper lung field Opaque medium puddled in saccular dilatations of diseased bronchi in right lower field Similar but less pronounced process at left base

X-Ray Findings

Like the physical signs, these depend to a large extent on changes in the lungs. There is no typical picture. The cylindrical form shows increased linear markings. Saccular ectases appear as ring shadows with or without a fluid level; they are sometimes more clearly demonstrable by comparison of films taken *before and after evacuation of secretion* by cough The presence and extent of bronchial dilatation can be more accurately determined by films taken after injection of contrast medium

Diagnostic Pitfalls

Chronic Bronchitis. *The frequency with which chronic bronchitis and bronchiectasis* coexist has already been mentioned. Bronchiectasis is probably present if a patient with chronic bronchitis produces large amounts of the characteristic sputum or develops signs of pleuropulmonary fibrosis, or clubbed digits

Tuberculosis. Chronic cough, expectoration, especially if bloody, and presence of abnormal pulmonary signs frequently cause bronchiectasis to be erroneously diagnosed tuberculosis or, sometimes, *vice versa* When the two diseases coexist, one or the other may be overlooked. Involvement of the apices is likely to indicate tuberculosis, of the bases, nontuberculous bronchiectasis, but this rule is by no means infallible. Differential diagnosis depends largely on x-ray and repeated examination of sputum and gastric contents.

Abscess of Lung. Onset is usually acute. A history of recent acute illness or of recent operation in the mouth or upper respiratory tract is the rule. *In bronchiectasis the process begins insidiously. X-ray studies and bronchoscopy are* important

P[illegible] [illegible] followed by [illegible] are likely The important [illegible] and abrupt onset of profuse purulent expectoration Microscopic examination of the sputum in perforating empyema usually shows pneumococci or, less often, streptococci alone. The sputum of bronchiectasis contains mixed flora. X-ray is essential.

Pulmonary Gangrene. The onset is rapid, usually occurring following pneumonia or pulmonary infarct. The chronic cases are very difficult to distinguish from bronchiectasis, but if elastic fibers can be demonstrated in the sputum, the likelihood is that abscess or gangrene, not bronchiectasis, has developed. The *foul odor of gangrene is characteristic.*

Bronchial Obstruction

Partial or total obstruction of a large or small *bronchus is created by the following causes.*

1. Tenacious secretion complicating bronchopulmonary infection.
2. Disease of bronchial wall such as a tumor, tuberculosis, or a non-specific inflammatory process.
3. *Foreign body.*

4 Pressure from without by tumor, aneurysm, enlarged lymphnodes, or marked dilatation of an auricle

The symptoms and signs which depend on the secondary changes in the lung vary with the acuteness of onset, size of the bronchus involved, and degree of obstruction An entire lung, a single lobe, or a smaller area may be affected. *If obstruction is suspected, bronchoscopy and perhaps bronchograms are essential.*

Symptoms

There are no symptoms which can be called peculiar to obstruction. When it occurs *suddenly* convulsive cough is prominent and the patient may have a sensation of respiratory restriction or local thoracic discomfort. Involvement of a large bronchus will cause dyspnea and perhaps a feeling of suffocation. When obstruction develops *gradually*, persistent and nagging cough is the rule. Wheezing, of which the patient is aware and can sometimes localize, is likely Occasionally he will report a sensation of restricted breathing or dull pain on the affected side. Constitutional symptoms are dependent on the cause and on secondary infection in the involved lung resulting from impaired drainage.

Signs

The local signs denoting successive stages in the course of gradual obstruction appear in chronological sequence as indicated below Obviously, following sudden obstruction, the signs will reflect the degree of ventilative impairment

No Signs.

Wheezing Respiratory Sounds. These are heard over a limited area of the thorax The patient may be aware of them

Localized Emphysema. Usually accompanied by more pronounced local *wheezing, this develops when obstruction is sufficiently* advanced to cause trapping of air, which, in turn, is dependent on the fact that air flows into the *lung during inspiration more readily than it is expelled during expiration* Valvular action of the obstructing lesion may also be operative If the emphysematous area is large enough to be clinically detectable, one will find a limited area of increased resonance, diminished breath and voice sounds, and absent tactile fremitus Occasionally, the inspiratory sound in the affected area is delayed As the patient inhales, the thorax expands normally, but no breath sound is audible over the affected region until almost the end of inspiration, whereupon a sudden inflow is heard X-ray may show localized emphysema which is not clinically evident

Indications of Complete Obstruction. When no air reaches lung parenchyma, the affected area will show.

1 Dulness to flatness
2 Absent tactile fremitus
3 *Greatly diminished to absent voice and breath sounds*
4 Displacement of mediastinal contents toward the affected side. This occurs only with obstruction of a fairly large bronchus.



[illegible] largement of heart shadow due to coincidental arteriosclerotic heart disease and previous myocardial infarction (Courtesy Dr Egon G Wissing, Chief, Radiology Service, Boston Veterans Administration Hospital)

Indications of Infection. *Due* to impaired drainage, the segment of lung distal to the obstruction will become secondarily infected This will be manifested by fever, perhaps increase of cough, and moist rales or signs of consolidation over the affected area Rarely cavitation can be identified These changes often appear before obstruction is complete

X-Ray Findings

In partial obstruction, the affected segment of lung, since it contains trapped air, appears as an area of increased aeration on the expiratory film If the lesion *involves a major bronchus, the heart and mediastinum tend to shift slightly* toward the affected side on inspiration and away from it on expiration Properly penetrated films may show actual narrowing of the bronchus When obstruction becomes complete, air-containing bronchus beyond the point of narrowing cannot be seen, the distal segment of lung will be atelectatic, and shift of mediastinum is more pronounced. In either partial or total obstruction, infection will be indicated by parenchymal infiltration with or without abscess formation.

Foreign Body Inhalation

Foreign body inhalation and bronchogenic tumor as causes of bronchial obstruction merit special mention *Although sometimes encountered in adults,*

the former is more common in children. It is likely to be overlooked because of inadequate history and should always be suspected when a respiratory tract ailment does not conform to one of the usual patterns.

At onset there is convulsive cough, gagging, and a sense of suffocation lasting a few hours. After a latent period of two or three days, the secondary manifestations appear. In the acute case, they simulate those of pneumonia or abscess, with cough, purulent, often foul, sputum, and varying degrees of *continuous or intermittent fever*. in the more chronic case, the symptoms suggest asthma, tuberculosis, or bronchitis If the offending agent has lodged in a small bronchus, the primary symptoms may be entirely absent and secondary manifestations may be the first hint of trouble. Because the picture is then so similar to that of pneumonia, tuberculosis, or perhaps asthma, and a foreign body is apt to be radioparent, the diagnosis is often overlooked If x-ray fails to show foreign material, its presence would be suggested by *demonstration of obstructive emphysema* by comparison of films taken at full inspiration and full expiration (*see* Chap 27).

Tumor of Bronchus

When persistent cough, particularly if productive of scanty, bloody sputum, develops in a previously healthy adult, bronchogenic tumor must always be suspected, especially if tuberculosis has been ruled out. The same applies if x-ray or physical examination demonstrates a local lesion not readily explained on some other basis

Tumor of the bronchus follows the clinical course of slowly developing bronchial obstruction described above.

PNEUMONIA

LOBAR PNEUMONIA

The term *lobar pneumonia* refers to a specific acute infectious disease involving part or all of one or more lobes of one or both lungs. It is distinct from *bronchopneumonia*, a malady in which small isolated or large confluent patches of disease are scattered through one or both lungs Lobar pneumonia is a *primary* disease, bronchopneumonia, usually a *secondary* process complicating some infectious or debilitating malady The former is almost always caused by the pneumococcus, rarely by Friedlander's bacillus Pneumonia showing lobar distribution clinically is sometimes caused by *the streptococcus or staphylococcus*, but pathologically these cases are usually found to be confluent bronchopneumonia

Since the introduction of the sulfonamids and antibiotics, lobar pneumonia appears to have diminished in frequency and, thanks to early initiation of treatment, rarely pursues a course similar to that which pertained previously. The apparent diminished morbidity can be attributed in part to interruption *of the disease so early that it is not recognized as such The picture, as described* below, represents the course to be expected only when specific therapy is started late or is, for some reason, ineffectual.

Symptoms

Onset is strikingly sudden, it may be preceded by upper respiratory infection or appear without any hint of previous trouble

Systemic Response. *High fever, malaise, and prostration appear* early. The disease is often ushered in by a single chill. If the patient has more than one chill within the first day or so, lobar pneumonia is unlikely, although a subsequent chill may herald involvement of another lobe

Cough. This is usually productive of thick, viscid sputum which is raised with difficulty In the early stage it is frankly bloody or, due to mixture of blood with mucoid material, rusty, later it becomes yellowish and mucopurulent

Pain in Chest. Experienced on the affected side and increased by deep breathing or cough, this is often excruciating It is due to coexistent fibrinous pleuritis If the diaphragmatic pleura is involved, the pain may be referred to the abdomen and lead to a mistaken diagnosis of gallbladder disease or, especially in children, acute appendicitis

General Signs

In all but the mildest cases, a look at the patient will show:

Characteristic Facies. The skin is flushed, expression anxious, and the alae nasae move with respiration Herpes of the lips or some other part of the face is likely

Distressed Breathing. Respiration is rapid, obviously painful, and, because of pleural pain, likely to be jerky and accompanied by a characteristic expiratory grunt.

Cyanosis. This is usually not pronounced unless the patient is excessively toxic or a large portion of pulmonary parenchyma is diseased

Pulmonary Signs

Signs referable to the lungs vary with the location, extent, and duration of the process For the first day or so they may be altogether absent A small patch of solidification deep in the lung produces no signs at the periphery; one near the surface may be too superficial to create changes appreciable on percussion or auscultation Later if the pneumonic area enlarges, signs will appear but *a case of lobar pneumonia may run its entire course without producing any signs in the lungs* Diagnosis then depends on the history, x-ray, and laboratory findings. Since specific therapeutic agents are much more effective when administered early, recognition of the disease on the basis of the general clinical picture prior to the advent of local signs is highly important

The early signs are those of solidification described below but they are by no means so clear-cut, and often can be detected only by the most meticulous examination. Impaired resonance may be demonstrable only by comparison of the two sides The breath sounds are likely to be diminished and bronchovesicular rather than bronchial, and spoken voice sounds show only slight alteration Change of whispered voice is often the most dependable early indication of the pneumonic process, its expiratory phase is somewhat prolonged and higher in pitch than normal, intensity diminished, normal or increased If there is an associated acute pleuritis, one may hear a friction sound, but often it is not apparent because of restricted respiratory movement due to pain When heard in a case presenting the general clinical picture of beginning lobar pneumonia but without evidence of solidification, it is most suggestive Early signs of upper lobe involvement are likely to appear first at the apex of the axilla, abnormal sounds will be overlooked unless one pushes his stethoscope as high as possible into this region

When the pneumonia is well-established and a large portion of a lobe has become infiltrated, the typical signs of solidification, as indicated below, are present It must be emphasized, however, that current specific therapy often arrests the disease before this picture has time to develop

Increased Tactile Fremitus. Although usually increased, fremitus occasionally is decreased or absent as a result of marked acute or chronic pleural thickening

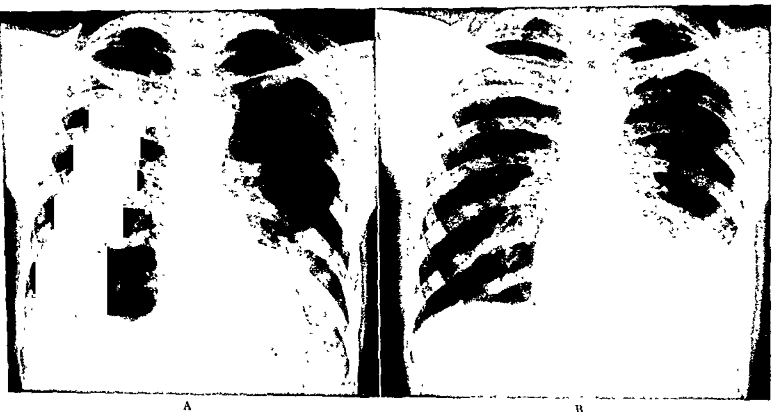


Fig 25 1 Lobar pneumonia

A Two days after onset Small area of hazy density at left base

B One week later Homogeneous dense shadow involving lower third of left lung field.

Dulness. The percussion note is obviously dull, sometimes almost flat.

Bronchial Breath Sounds. Usually these are loud and seem close to the ear; rarely, they are diminished

Bronchial Whispered Voice Sounds. These are usually intense. Testing the whispered voice is especially useful when breath sounds are faint or the patient cannot breathe deeply because of cough or pleural pain.

Bronchial Spoken Voice Sounds. Appearing loud and close to the ear, the voice sounds are usually bronchial but sometimes have a nasal or bleating quality (*egophony*). The latter is more characteristic of fluid in the pleural cavity, especially when it follows pneumonia.

Rales. Medium or fine rales are heard during inspiration except when the air passages become filled with exudate. They reappear during resolution as coarser sounds which sometimes have a consonating quality.

Pleural Friction Sound. This may or may not be present.

Occasionally, when signs of solidification are pronounced at one base, *similar signs will be found at the other adjacent to the spinal column* These are merely transmitted from the diseased side and only when extensive do they denote actual involvement of the other lung

At the height of the illness, especially in a serious case, physical signs described above may be entirely altered by plugging of a large bronchus with exudate. The percussion note becomes very dull to flat. Tactile fremitus, breath and voice sounds are diminished or absent, and rales disappear. This picture can remain unchanged for several days and, since it closely resembles that of fluid, often leads to a mistaken diagnosis of pleural effusion or empyema. When the exudate is dislodged by cough or absorbed, the typical signs of solidification reappear

X-Ray Findings

The earliest manifestation is a central density extending out from the hilus in a fan shaped manner, or a peripheral density extending medially. The process is quite homogeneous but with ill-defined margins As the disease advances, the consolidation spreads to involve most or all of one or more lobes. Regression begins with patchy areas of radiolucency appearing in the consolidation and progresses until the process has cleared entirely Since the advent of specific therapy, the classic x-ray picture of lobar pneumonia is rarely seen; when it is, clearing of the lesion as indicated by x-ray lags behind improvement of systemic changes

Course

Prior to the advent of specific therapy, uncomplicated lobar pneumonia lasted from 3 to 10 or 12 days, rarely longer. It often terminated by *crisis* within 12–24 hours, the temperature, pulse, and respiratory rate dropped suddenly from a high to a normal or almost normal level, and the patient's general condition showed striking improvement In other cases recovery was by



FIG 25 2 Lobar pneumonia, right upper lobe Homogeneous density involving all but apical segment

lysis Resolution started when the febrile reaction began to subside Solidification usually disappeared entirely within 1–4 days, only occasionally would it take as long as a week or more. During this stage moist rales, usually coarse and bubbling, were present.

Current treatment, if initiated early, often arrests the process before it has fully developed The febrile and constitutional responses are expected to subside within 3–4 days but it is striking that changes in the lung, if well-established, disappear certainly no more rapidly and sometimes more slowly than in cases of spontaneous recovery.

The disease occasionally runs an atypical, subacute course. It may spread from one part of the lung to another, or isolated patches of solidification will crop up one after the other in separated areas When it occurs in aged or debilitated patients, the characteristic manifestations may not be as clear-cut as in a younger and more vigorous person Local signs appear in conjunction with remarkably mild systemic reaction or ill-defined local signs with a typical systemic response.

The leukocyte count is almost always elevated—14,000 or more—a finding of importance in differentiating between lobar pneumonia and atypical pneumonia, pulmonary tuberculosis, pleurisy with effusion, or influenza, in all of which the white count is at or near the normal level. Except in a mild case, a persistently low leukocyte count in lobar pneumonia is an unfavorable sign

If abnormal signs persist for more than a few days after the acute process appears to be over, some complication such as empyema, abscess, or pleural effusion is likely. Slow resolution or persistent atelectasis from bronchial obstruction may be the cause, but the diagnosis of either should always be set aside until the above-mentioned complications have been excluded. Other

complications are acute fibrinous pericarditis, acute otitis media, pulmonary gangrene, pneumococcus endocarditis, and, due to rupture of a small pleural abscess, spontaneous pneumothorax followed by pyopneumothorax. Especially in the aged or debilitated, purulent parotitis is sometimes encountered.

Diagnostic Pitfalls

Serous Pleural Effusion. Dulness and diminished or increased bronchial breath sounds heard over fluid can lead to a mistaken diagnosis of pneumonic solidification, particularly when onset of the effusion is acute. The significant points of difference are:

1. Over fluid, the percussing fingers encounter a greater sense of resistance than over an area of solidification, except when the lung is unusually solid, as in Friedlander's infection.
2. Over fluid, rales are rarely heard; over solidification, they are usually present unless no air is getting into the region.
3. If tactile fremitus is found, fluid is not present. The converse is not true, for fremitus is sometimes absent or much diminished over solidification or atelectasis.
4. Over fluid, egophony is usually heard; over consolidation, it is less common.
5. Motion on the affected side is more restricted with fluid than with consolidation.

If a bronchus leading to a pneumonic area is plugged, the physical signs more closely simulate those of fluid. The important differential point is that, with fluid, the heart and mediastinal contents are apt to be *displaced away* from the affected side, whereas with solidification and bronchial obstruction they are either *not displaced* or *displaced toward* the affected side. Furthermore, in pneumonia and bronchial obstruction, sudden cough may dislodge the plug and restore the typical pneumonic picture, with fluid no such sudden change occurs. Differences in onset, course, appearance of the patient, leukocyte count, sputum and x-ray are also pertinent. Sometimes physical and x-ray findings fail to differentiate between the two conditions, whereupon exploratory thoracentesis must be utilized.

Purulent Pleuritis (Empyema). (*See* Chap. 28.)

Bronchopneumonia. Bronchopneumonia is secondary to some other disease, pursues a different course, and shows different physical signs. Difficulty is most likely to arise when the bronchopneumonia, being confluent, shows large areas of solidification in one or more lobes. This form is most apt to occur in children, the aged or debilitated and in cases of streptococcus or staphylococcus infection. Differentiation may be clinically impossible.

Acute Tuberculous Pneumonia. In the early stages this cannot be distinguished from lobar pneumonia. Onset is acute, with rapid rise in temperature, pain in the side and, sometimes, a chill. The typical signs of pulmonary solidification are found. A previous history of tuberculosis may be misleading, for a patient with inactive or mildly active tuberculosis can develop an attack of

pneumococcus lobar pneumonia. It is characteristic for the leukocyte count to be normal or *somewhat lower than normal in tuberculous pneumonia*, yet it is sometimes as high as in pneumococcus pneumonia, and the latter occasionally shows a low leukocyte count. In the earlier stages of tuberculous *pneumonia*, tubercle *bacilli* may not be present in the sputum. After 10 or 12 days this disease can usually be recognized by the following:

1. Recovery does not occur promptly, as in lobar pneumonia. Fever continues, shows wider range and, unless one of the newer anti-tuberculous agents is effective, *the patient steadily loses ground.*

2. Profuse amounts of sputum containing large numbers of tubercle bacilli are expectorated.

3. When areas of softening develop, x-rays are characteristic.

Tuberculous pneumonia should be suspected in any case which appears to be lobar pneumonia but which does not respond to the usual treatment *Repeated examinations of the sputum are imperative.*

Pulmonary Embolism with Infarction. (*See* Chap 27). *Pulmonary infarction* is marked by fever, pain in the side, dyspnea, perhaps bloody sputum, and later often shows signs similar to those of a small patch of pneumonia Existence of obvious thrombophlebitis, phlebothrombosis, or some situation known to encourage either, such as local trauma to a lower extremity, congestive failure, recent operation or labor, prolonged immobilization, or cancer, especially of the pancreas, favors embolism Pleural pain, often the first symptom of infarction, appears abruptly. Following operation or labor, trouble starting within the first two or three days is more likely to be pneumonia, appearing later, infarction. A large infarct causes sudden collapse, extreme dyspnea and cyanosis *The pulmonary signs of infarction are likely to disappear* more quickly than those of pneumonia

Compression Atelectasis. The signs overlying a portion of compressed lung simulate those of solidification. A large *pericardial effusion, extreme cardiac* hypertrophy, or pronounced dilatation of the left auricle may cause compression in the left midback; any intra-abdominal-variant, such as ascites or pregnancy, which pushes the diaphragm upward will compress one or both bases. When the signs are due to compression, the typical clinical picture of pneumonia will be absent and one will find, clinically or by x-ray, a cause for the compression

Obstructive Atelectasis. (*See* Chap 27). Uncertainty between lobar pneumonia and obstructive atelectasis is most likely to occur in the postoperative case The latter, usually occurring within a day or so after operation, is marked by *sudden onset of fever, cough, dyspnea, and sometimes* pain in the chest. The severity of symptoms and extent of signs vary with the size of the area involved At onset one will find a zone of flatness, absent tactile fremitus and absent or *greatly diminished breath and voice sounds*, but within a day or so, they are followed by signs indistinguishable from those of solidification The latter may disappear in a few hours, rarely lasting more than 3 or 4 days. The most im-

portant differential point is that with appreciable obstructive atelectasis, physical examination or x-ray will show the heart and mediastinum to be displaced *toward* the affected side; by palpating in the suprasternal notch one may find that the trachea has also been drawn over. In pneumonia and infarction, these structures are not displaced or are very slightly moved *away* from the affected side.

Acute Intra-Abdominal Disease. Lobar pneumonia of the right lower or middle lobe is sometimes confused with acute appendicitis or acute cholecystitis, because of the tendency of associated pleural pain, especially when the diaphragm is involved, to be referred to the abdomen. In children it is quite common for beginning right-sided lobar pneumonia to be diagnosed acute appendicitis. The reverse is rarely true, acute appendicitis is seldom diagnosed lobar pneumonia. Any acute infection in children is apt to be ushered in by nausea and vomiting and when, in addition to these symptoms, there are pain in the right side and no demonstrable signs in the lungs, the error is easily made. The differential diagnosis is made on the different general manifestations of the two diseases. The patient with lobar pneumonia usually has a more severe systemic reaction than one with appendicitis, and his temperature, pulse, respiratory rates, and leukocyte count are higher. *Any case of suspected appendicitis in a child should, particularly if the systemic reaction is severe, be studied with lobar pneumonia in mind.* Careful x-ray examination may be the only means of establishing early diagnosis and preventing unnecessary operation.

Acute Cerebrospinal Meningitis. In children, the onset of lobar pneumonia is occasionally attended by headache and signs of meningeal irritation (*meningismus*). Examination of the spinal fluid may be necessary to exclude meningitis, unless the physical or x-ray evidence of lobar pneumonia is unequivocal.

BRONCHOPNEUMONIA

Bronchopneumonia is inflammation of the terminal bronchioles and the surrounding lobules, a process which results in the formation of small, discrete areas of solidification distributed more or less throughout the lungs, but found chiefly in dependent portions. The patches may be small and scattered or may coalesce to form larger areas like those in lobar pneumonia. The disease is secondary, occurring as a complication of some respiratory infection such as acute coryza, bronchitis, or influenza, of certain specific infectious diseases, especially measles, whooping cough, or diphtheria, or of acute sepsis. It is a frequent terminal complication of congestive heart failure, nephritis, malignant and other chronic or wasting diseases. It may develop following anesthesia or inhalation of irritating materials, such as dust or gas. In contrast to lobar pneumonia, there is no specific etiologic agent. Any of the common pathogenic organisms is capable of causing the disease. Mixed infection is frequent.

Occasionally in adults, but quite commonly in the young, especially infants, one encounters a fulminating form of bronchopneumonia usually due to streptococcus, staphylococcus or pneumococcus, which shows pulmonary changes

clinically indistinguishable from those of unilateral or bilateral lobar pneumonia. In infants the disease may be so rapidly fatal that it leads to a mistaken diagnosis of death from suffocation in the crib.

Symptoms

The manifestations of ordinary bronchopneumonia are so varied that there is no one typical picture Its symptoms, signs, and course are influenced by several factors, the chief of which are the age of the patient, virulence of the infecting organism, extent of the pulmonary involvement, and nature of the antecedent disease. Its recognition often depends as much on its general manifestations as on the changes in the lungs, for small bronchopneumonic patches often exist without giving rise to the characteristic signs of solidification

The appearance of bronchopneumonia as a complication of an already existing disease is sometimes insidious, sometimes rapid. When the antecedent disorder is respiratory, such as whooping cough or bronchitis, bronchopneumonia manifests itself as aggravation of the existing systemic and respiratory symptoms. Cough and respiratory distress become pronounced, cyanosis may appear, and the temperature, pulse, and respiratory levels rise When the antecedent disease is other than respiratory, systemic symptoms become aggravated and new symptoms, such as cough and dyspnea, appear. Cough is usually productive of yellowish mucopurulent or purulent material which is occasionally blood-tinged The typical rusty sputum of lobar pneumonia is rare.

Signs

Except in extremely debilitated patients who may show surprisingly little elevation of temperature, there is irregular fever varying from 102° to 104° or higher The pulse rate varies from 100 to 140, and the respiratory rate from 20 to 40 or higher. In severe cases the indications of toxemia are pronounced, and respiratory distress and cyanosis are prominent.

The local signs are as variable as the symptoms They appear in many combinations Sometimes there are no characteristic physical findings; they may have been altered or masked by those of the underlying disorder such as bronchitis, emphysema, bronchiectasis, or congestive failure on which the pneumonia is superimposed In the fulminating form mentioned above, one finds marked systemic reaction, prostration, dyspnea, cyanosis, and high fever. Local signs similar to those of lobar pneumonia may be found, but sometimes, especially in infants and debilitated adults, the infection progresses with such rapidity that death ensues before they have time to develop Here the diagnosis is made on the basis of the general picture In the ordinary case of bronchopneumonia one will usually find

Rales. Typically these are fine or medium and moist They may be generally distributed or limited to one or more areas, usually the bases When there is a coexistent bronchitis, the finer, more moist rales of the pneumonia may be masked by the coarser bronchial rales.

Signs of Solidification. These show wide variation, due to diversity in size

and density of the pneumonic patches. Smaller patches may produce no signs at all. Those of moderate size modify the resonance, fremitus and breath sounds in much the same way as lobar pneumonia, except that the changes are usually less pronounced, and the borderline between diseased and healthy parenchyma is not well-defined. Large confluent areas of infiltration may show signs altogether indistinguishable from those of lobar pneumonia. Except in the confluent type, the signs are likely to change from day to day as a result of resolution of certain areas, involvement of new areas, or variations of ventilation secondary to plugging or unplugging of bronchi.

X-Ray Findings

These vary with the character and extent of the pathologic changes. The typical case shows irregular patches of increased density less homogeneous than in lobar pneumonia Small patches may become confluent; involvement of several lobes is likely.

Diagnostic Pitfalls

Acute Bronchitis. The important clinical feature which distinguishes bronchopneumonia from the acute or chronic bronchitis which it often complicates is the aggravation of the respiratory symptoms and the systemic reaction. Small pneumonic patches may or may not be demonstrable by physical or x-ray examination

Lobar Pneumonia. The important differential points are the mode of onset, character and distribution of the lesions, course of the disease, and often the bacteriologic findings Differentiation between lobar and confluent bronchopneumonia may be clinically impossible. If *Streptococcus hemolyticus* predominates in the sputum the case is probably bronchopneumonia.

Tuberculous Bronchopneumonia. This is most likely to involve the apices and upper lobes, while ordinary bronchopneumonia favors dependent areas; its course is longer and featured by greater loss of weight and strength. Tubercle bacilli are usually demonstrable in the sputum

Pleural Effusion Atelectasis of a lower lobe occurring in bronchopneumonia from plugging of a bronchus may give signs resembling those of pleural effusion The differential features are virtually the same as between pleural effusion and lobar pneumonia. In fulminating confluent bronchopneumonia, especially when due to streptococcus, purulent fluid may accumulate in the pleural cavity within the first few days and so obscure the signs of solidification that pneumonia is not suspected

Pulmonary Edema. Due to impaired circulation in the pulmonary circuit and found most often in left ventricular failure or tight mitral stenosis, pulmonary edema is characterized by moist rales, *especially at the* bases, and sometimes dulness, and diminished breath sounds which cannot be distinguished from those of bronchial pneumonia. Fever and other constitutional symptoms of infection are absent. Bronchopneumonia superimposed on edema of the lung will be indicated by such significant changes as increase in cough, eleva-

tion of temperature and leukocyte counts, general manifestations of infection and, possibly, more pronounced local signs.

Pulmonary Embolism with Infarction. Multiple small pulmonary infarcts may cause a general clinical picture and local signs in lungs often erroneously diagnosed bronchopneumonia. If the possibility of infarction is borne in mind, especially when there exists a background for pulmonary embolism, the correct diagnosis should be made. X-ray may be helpful.

PRIMARY ATYPICAL PNEUMONIA

During the last 2 to 3 decades, this form of pulmonary infection, rarely encountered previously, has become prevalent in all parts of the world. Etiology is unknown, but as a result of extensive epidemiologic studies, authorities agree that virus infection is responsible. It is highly probable that several different agents are at fault; with one possible exception, no specific virus has been isolated. Although the disease has a predilection for the young and middle-aged, no age group is exempt. It is likely to occur in epidemics in homes, school, and other institutions The morbidity rate was high in military installations during the recent war. The picture as described below represents what might be called average; cases of lesser or greater severity are not infrequent Often a patient will continue at his work with symptoms which are regarded as nothing more than a "bad cold." Here diagnosis is likely to be overlooked unless by chance an x-ray is taken. Sometimes the disease is severe, the patient remaining ill for weeks.

Symptoms

Acute upper respiratory infection may or may not precede onset. The latter may be insidious or acute, with feverishness, chilliness, but not true chill, cough, and perhaps substernal discomfort. Pleural pain is rare Headache, backache and bone aches are often striking Perhaps the most outstanding symptom is harassing, persistent cough which is worse at night and not controllable by ordinary remedies It is usually dry but, in the late stages, may be productive of mucoid or mucopurulent sputum

Signs

As a rule the patient appears no more ill than one with acute upper respiratory infection He is not toxic and respirations are neither rapid nor labored. Temperature is variable and irregular, although it may reach as high as 103°–104° it is rarely sustained at such a high level. However, it is likely to be out of proportion to the general appearance of the patient and other manifestations of trouble.

Another outstanding feature is the paucity of physical signs, especially when compared with the x-ray evidence of pulmonary disease During the early stage, lungs may appear perfectly normal Sometimes one may discover an area of slight dulness or of diminished breath sounds, or a few moist or sibilant rales Often these minimal changes constitute the only local evidence of disease even



FIG 25.3 Primary atypical pneumonia, bilateral Patchy, poorly demarcated area of density at right base, linear shadows at left base No abnormal physical signs detected

when by x-ray pulmonary infiltration is found to be extensive. Later, definite dulness, perhaps with slight alteration of breath sounds and abundant moist rales may be found, but many cases run their entire course without any well-defined local signs Bronchial breathing and changes in whispered or spoken voice are rare

X-Ray Findings

Hazy areas of ill-defined density in one, sometimes more than one, lobe are the rule. Rarely, in a severe case, a miliary process may be observed or extensive consolidation which cannot be distinguished roentgenologically from that of lobar pneumonia

Depending on its severity, the disease lasts from 1 to 3 or 4 weeks, with gradual improvement and slow convalescence In contrast to lobar and bronchopneumonia, leukocyte and differential counts are normal In the early stage, sputum is scanty and shows no predominant organisms such as one finds in lobar pneumonia Later, especially if secondary bronchitis has developed, a moderate amount of mucopurulent sputum will be coughed up In the serious form fever, severe headache, cough and prostration are pronounced, but even here signs in lungs are likely to be minimal Although recovery is the rule, death occasionally occurs Antibiotic or other specific therapy is ineffective. Complications are not common Pleural effusion sometimes occurs, and if it does, may accumulate with rapidity requiring two or more thoracenteses before the

process quiets down Occasionally bronchiectasis will develop but more often signs and x-ray findings suggesting bronchiectasis are due to slow resolution and, given time, will disappear.

Diagnostic Pitfalls

As a rule, distinction between lobar or bronchopneumonia and atypical pneumonia can be made on the basis of the general picture, physical signs, x-ray, and laboratory studies Occasionally, however, one cannot be certain, in such a case, the response to antibiotic therapy is highly significant. From upper respiratory disease one must rely on x-ray for orientation Some rickettsial diseases such as Q fever, coccidioidomycosis, and virus diseases such as influenza and psittacosis, may resemble atypical pneumonia and can be differentiated only by laboratory studies. If a case thought to be virus pneumonia persists for an unusually long time, the possibility of bronchiectasis or parenchymal infection secondary to bronchial obstruction from tumor or other cause must be excluded.

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PULMONARY TUBERCULOSIS

Although it is a widespread disease, tuberculosis in this country is showing diminishing incidence, presumably as a result of earlier recognition, improved

[illegible]

impairment and show no other local changes of importance in the lungs or elsewhere. Some clinics refer to cases in this group as having *tuberculous infection*, distinguishing it from *tuberculous disease*, in which the organism has gained a stronger foothold, involvement of the lungs is greater, or some other part is affected

Pulmonary tuberculosis appears in a number of forms, the pathologic changes showing marked variation in character and extent In general the lungs respond in one of two ways

1 An *exudative* reaction, in which constituents of plasma, and inflammatory cells mostly of the large mononuclear type, infiltrate the tissues without replacing normal structure Such a lesion may partially or almost completely resolve or progress to caseation The caseous lesion in turn may liquify and excavate into a bronchus, or become encapsulated, largely or completely fibrosed, and perhaps calcified.

2 A *productive* reaction, characterized by greater cellular infiltration which replaces normal tissue, and formation of tubercles. Following the latter the productive reaction may regress to non-specific scar formation, may encapsulate and slowly fibrose, or may progress to caseation with eventual excavation, fibrosis, or calcification.

Most cases represent a combination of these two processes, either of which may be predominant The systemic response is likely to parallel the pulmonary changes but this is not necessarily true It is possible for a patient with relatively little pulmonary change to show fairly severe constitutional reaction, while another with extensive pulmonary involvement will have little or no evidence of ill health

When constitutional symptoms are present, or there is evidence that the process in the lungs is progressing, the case is one of *active* pulmonary tuberculosis If there are no constitutional symptoms, if periodic physical and x-ray examinations reveal no advance of the pulmonary changes, and if bacteriologic

studies are negative, the case is called *inactive*. The terms must be used with caution, for in any person the systemic and local changes are capable of having remissions and exacerbations; it is always possible for one with little pathologic change to develop severe disease.

PRIMARY TUBERCULOSIS

The primary infection in tuberculosis can occur at any age. In this country at present, almost all cases are thought to represent contact infection from a patient with cough and positive sputum Presumably because of earlier recognition of the disease and better protection of children from infected parents and others, primary disease is becoming less frequent in the young, relatively more so in adults.

As a rule, the initial lesion, situated anywhere in the lung parenchyma but usually not far from the periphery, takes the form of a small bronchopneumonic patch which may become necrotic It is usually single, occasionally multiple. By lymphatic spread, the regional bronchial nodes become enlarged and necrotic, this feature is more pronounced in the young than in adults. The *primary complex* (parenchymal and lymphnode lesions) is often so small as to be undetectable either by physical examination or x-ray. During the active stage, symptoms may be entirely absent or there may be listlessness, malaise, easy fatigability and, in the child, failure to gain weight Cough is inconsequential. When the complex is perceptible, one will see by x-ray a small patch of hazy density in the lung or enlarged peribronchial lymphnodes, occasionally both If these are absent, the only evidence of tuberculous infection is a positive tuberculin reaction which develops usually between 4 and 6 weeks after exposure.

In a certain number of cases, pulmonary change, lymphnode enlargement, or both, are quite evident by x-ray but only when they are unusually pronounced will physical signs of trouble be detectable In the latter event one might find evidence of pulmonary infiltration, or mediastinal lymphnode enlargement. Even when parenchymal or lymphnode involvement is quite pronounced, systemic response is not necessarily striking, although listlessness, loss of weight, and low-grade fever are the rule.

Primary infection is sometimes indicated by the sudden appearance of *pleurisy with effusion* due presumably to extension from an imperceptible subpleural parenchymal lesion or direct transmission of organisms through the lymphatic channels from a terminal alveolus to the pleura. Any case of spontaneously developing pleural effusion, especially in an adolescent or adult, should be regarded as tuberculous until proved otherwise

Once primary infection has occurred, there are three possible outcomes:

1. In most cases the complex heals by fibrosis, encapsulation, and calcification. X-ray may then show a small calcified 0 5- to 2-cm. nodule in the lung parenchyma (*Ghon's node*) and perhaps calcification in the regional lymphnodes. Tuberculin reaction will remain positive for years.

2. In a small number of cases, the disease progresses by direct extension or

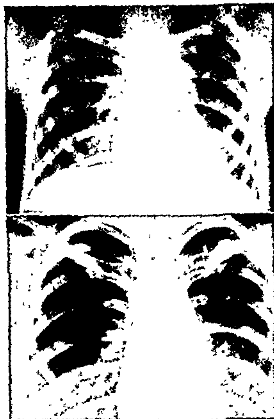


FIG. 261 Primary tuberculosis.

A Hazy density involving apex of left lung in a child age 3 Lobulated shadow at upper pole of left hilus represents enlarged lymphnodes

B. After 2 years of therapy Pulmonary infiltration has cleared, leaving a small calcified nodule (Ghon's tubercle) visible just above medial third of clavicle Lymphnodes have become smaller and calcified (Courtesy Middlesex County Sanatorium, Waltham, Mass)

intrabronchial spread, by rupture of a diseased node into a bronchus, causing tuberculous pneumonia, or by lymphatic or blood stream dissemination from the parenchymal or nodal component. The last-named will create generalized miliary tuberculosis or seeding in various organs which may or may not end in one or more of the complications listed below

3. The complex, not healing completely, lies dormant for some time but sooner or later its pulmonary or nodal component again becomes active and causes trouble in one of the following ways:

a The pulmonary or lymphnode focus, by rupturing into a bronchus, creates tuberculous foci in other parts of the lungs (*endobronchial dissemination*).

b. Invasion of a blood or lymph vessel causes further trouble in the lungs or, by distant seeding, creates disease such as cutaneous tuberculids, lymphnoditis, meningitis, renal, adrenal, genital, bone, gastrointestinal, or peritoneal

tuberculosis, or generalized miliary tuberculosis (*hematogenous or lymphohematogenous dissemination*).

PROGRESSIVE TUBERCULOSIS

As just noted, this may result from direct progression of primary disease or from reactivation of a primary complex which has lain dormant for some time. Serious disease in the child is usually the result of the former. In the adult, progressive tuberculosis is more likely a manifestation of reactivation (*reinfection tuberculosis*) although often, especially in adolescents and young adults, it is a new infection from without. The form the disease takes in the lungs depends on several factors: the manner in which infection has extended from the primary lesion or new portal of entry, the severity of the dose, virulence of the infecting organism, and sensitivity of the person to the tuberculous antigen. Infection may spread by way of the bronchi, lymphatics, or blood stream. Bronchial infection results from rupture of a lesion into a bronchus, from blood stream infection, or from direct invasion or extension from lymphatics.

Because of the diversity of its forms and the fact that the disease in each patient is undergoing continual change, it is difficult to devise any classification which will be both simple and complete. The newer forms of therapy have further complicated the classification problem.

Reinfection tuberculosis may show a response which is chiefly fibrinoserous with some cellular reaction, but no destruction of pulmonary tissue The lesion may be absorbed within a comparatively short time and leave no trace, if this occurs within less than 10 to 12 weeks, tuberculosis cannot be diagnosed with certainty unless tubercle bacilli have been found in the sputum or gastric secretion In some cases, the cellular element is greater, recovery is slower, and

losis In either case the development of tuberculous pneumonia or blood stream dissemination is possible.

Since impairment of health is not dependent solely on the extent of the pulmonary lesion, the status of a given case can be properly determined only by careful evaluation of the entire picture. constitutional reaction, character and extent of pulmonary lesion as observed on physical examination and x-ray, evidence of disease of other structures, isolation of tubercle bacilli from sputum or gastric secretion, and response to treatment as *revealed by periodic surveys*

SYMPTOMS

The subjective manifestations of active pulmonary tuberculosis can be divided into two groups (a) constitutional, and (b) focal. There is no particular combination peculiar to any particular form *but in general, the more advanced or acute the disease, the more severe* and numerous its symptoms. Any of those listed below may or may not be present.

Onset is often insidious In early and perhaps moderately advanced tuberculosis, the patient may feel well Because of this, many cases are not diagnosed until they have reached an advanced stage and the chances for recovery have become much less than would have pertained had the disease been recognized earlier Since x-ray is by far the most reliable detector of the early case, mass surveys, pre-employment, military, and similar examinations have, in recent years, brought to light many early cases which, thanks to prompt treatment, have been restored to good health. It is safe to say that otherwise many of them would have progressed to the point of serious trouble before being discovered. Sometimes, because of cough, malaise, and perhaps mild fever, the patient is at first thought to have an unusually persistant upper respiratory infection, or bronchitis In other cases, especially acute tuberculous pneumonia, onset is sudden, with high fever, cough, and prostration

Anything in the history suggesting the possibility of tuberculosis demands investigation known contact with a tuberculous person, poor hygienic surroundings, malnutrition, or occupation involving pulmonary health hazard Significant also is a history of a past illness which might have been of tuberculous origin, such as pleurisy with effusion, cervical lymphnoditis, chronic joint disease or ischeorectal abscess

Constitutional Symptoms

Fatigability. General malaise, easy fatigability, and weakness develop gradually, beginning as increasing difficulty in completing the normal day's work and, in the advanced case, reaching prostration

Fever. An important index of activity, fever is rarely over 99°–100° in the early or mild case It usually appears only in the afternoon, may be increased by exertion and will often disappear after a few days' rest Later it increases, has wider daily swings and considerable variation from day to day or week to week Chilly sensations often appear during the upward swing

Night Sweats Moderate sweating, especially at night, may occur in the earlier stages, drenching night sweats are common in the advanced case.

Anorexia. Loss of appetite or vague indigestion often appears early Loss of weight is likely Nausea, vomiting, and diarrhea occur in the very ill

Suppression of Menses. The periods may at first occur less frequently and be more scanty than normal, then disappear entirely

Focal Symptoms

Cough. Persistent cough always suggests tuberculosis, it is often the first manifestation At the start it is usually dry and hacking, later it is productive

Hemoptysis. This may vary from expectoration of slightly blood-stained sputum to frank hemorrhage Although a large number of cases of hemoptysis are due to tuberculosis, the symptom is by no means pathognomonic, it is often an important indication of some other bronchopulmonary disease such as bronchiectasis, tumor, or abscess, and of pulmonary congestion, especially in mitral stenosis It can also be caused by a local lesion in the mouth, nose or

throat. Frank hemorrhage from the lung is sometimes difficult to distinguish by history from hematemesis.

Thoracic Pain. Sharp pain, aggravated by deep breathing, is usually the result of associated fibrinous pleuritis or traction on pleural adhesions Dull soreness due to pleuritis or perhaps to pressure of enlarged lymphnodes sometimes occurs. Sudden, acute pain may reflect onset of pneumothorax. Pleuritis not associated with pneumonia or other acute respiratory infection strongly suggests tuberculosis or malignant tumor.

Hoarseness. Usually an indication of advanced disease, this results from complicating laryngeal tuberculosis Any case of hoarseness not definitely attributable to an acute upper respiratory infection, especially if it lasts more than a few days, demands careful investigation. Tuberculosis, syphilis, and cancer must always be excluded

Dysphagia. Also a late manifestation, painful swallowing indicates involvement *of the epiglottis, arytenoids, or other part of the mouth or throat.*

Chronic Caseous Tuberculosis

Also called *chronic ulcerative,* this is the most common type of progressive pulmonary tuberculosis Characterized by caseation, necrosis, varying degrees of fibrosis, and usually cavitation, it is a progressive disease, beginning almost always in the upper and dorsal portion of a lobe, most frequently an upper. The process gradually extends downward and if sloughing occurs, endobronchial dissemination brings about greater involvement of the same lobe and eventually of others As a rule, the upper part of the initially infected lobe shows further advanced lesions than the lower; one lung usually more disease than the other.

Although physical signs may be helpful in the diagnosis and evaluation of a case of tuberculosis, x-ray is much more important As stated earlier, a lesion too small to give physical signs will be detected by x-ray The lesion cannot always be specifically labeled by the roentgenologist but its discovery leads to further studies by which final diagnosis can usually be established. Furthermore, pulmonary changes can be more effectively followed by x-ray than by physical examination.

On the basis of the extent of the process, cases of pulmonary tuberculosis are classified as follows by the National Tuberculosis Association:[1]

Minimal. Slight lesions without demonstrated excavation confined to a small part of one or both lungs The total extent of the lesions, regardless of distribution, shall not exceed the equivalent of the volume of lung tissue which lies above the second chondrosternal junction and the spine of the fourth or body of the fifth thoracic vertebra on one side

Moderately Advanced. One or both lungs may be involved, but the total extent of the lesions shall not exceed the following limits

Slight disseminated lesions which may extend through not more than the volume of one lung or the equivalent in both lungs

Dense and confluent lesions which may extend through not more than the equivalent of one third the volume of one lung

[1] *Diagnostic Standards and Classification of Tuberculosis* New York, National Tuberculosis Association, 1955 Reprinted with permission of the National Tuberculosis Association.

Total diameter of cavities less than 4 cm
Far Advanced. Lesions more extensive than moderately advanced.

Minimal Disease

As already stated, early tuberculosis may exist without symptoms or there may be such complaints as fatigue, loss of weight, feverishness, chronic cough, or hemoptysis Physical signs are usually absent.

If the lesion is large enough, one may find, most likely at an apex, one or more of the signs indicated below. Examination must be carried out in an absolutely quiet room with the patient's arms folded across his lap, his shoulders lowered comfortably, and muscles well relaxed, in order to exclude adventitious muscle sounds. *Examination of the lungs during the 48 hours following an episode of hemoptysis or hemorrhage must be avoided because of the danger of provoking further bleeding Forced respiration or cough remain dangerous for several weeks*

Although both lung fields must be completely surveyed, special attention should be paid to the regions above and below the clavicles anteriorly, and above the scapular spines posteriorly. Auscultation should be performed first during normal respiration, then while the patient whispers, then during deep respiration. Following this he is asked to cough at the end of each expiration and then inhale briskly, the examiner must concentrate his attention on the initial phase of each post-tussive inspiration, the point at which rales are most apt to be heard In evaluating the percussion note, breath, and whisper sounds,

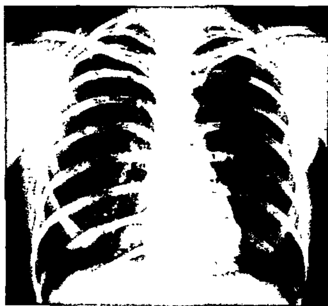


FIG 26 2 Minimal reinfection tuberculosis Small area of density in right upper lobe just below mid-third of clavicle On the film, small flecks of calcium representing the healed primary lesion are visible above the clavicle but have been lost in reproduction.

the modifications normally present at the apices must be kept in mind One looks for:

Slight Dulness.

Changes in Sounds. The breath sounds may be slightly diminished in intensity and show slight prolongation and higher pitch of expiratory phase. These variants may be more evident from the whispered voice.

Rales. Usually fine and crackling, rarely sibilant, these are localized, best heard at the end of inspiration, and frequently only during post-tussive inhalation Only rales which persist are significant.

Moderately Advanced Disease

General health is usually, but not necessarily, impaired; focal symptoms may or may not be present. Abnormal local physical signs may be absent but one or more of the following can usually be found, most likely at an apex:

Dulness. Dulness and a sense of increased resistance on light percussion is usually detectable. This change is sometimes best appreciated as narrowing of Kronig's isthmus

Alterations of Sounds. Bronchovesicular breathing and some increase in intensity of whisper sounds with slight prolongation and higher pitch of their expiratory phase are the rule

Rales. These may be similar to those sometimes heard over a minimal lesion or are consonating, sharper, and seem closer to the ear.

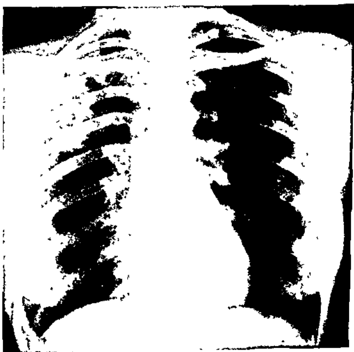


FIG 26 3. Moderately advanced caseous tuberculosis Cavity in right upper lobe and small area of infiltration toward periphery of left mid-lung field. (Courtesy Middlesex County Sanatorium, Waltham, Mass)

Restriction of Respiratory Motion. Due to parenchymal or pleural contraction, respiratory motion may be restricted and thoracic wall slightly retracted over the diseased segment.

Signs of Cavitation. Sometimes occurring in the moderately advanced but more frequently in the far advanced case, *cavities are often detectable only by x-ray.* Physical signs are not evident unless the cavity is large, has sufficiently rigid walls to prevent collapse, communicates directly with a bronchus, is situated near the lung periphery, and is not filled with secretion. It is likely to be overlooked in the morning and be detected later in the day after material which accumulated overnight has been coughed up. The most dependable signs are

AMPHORIC OR CAVERNOUS SOUNDS. Breath, whisper and perhaps voice sounds will have a hollow or cavernous quality. Surrounding these one may find a zone of bronchial sounds reflecting parenchymal infiltration

TYMPANITIC RESONANCE. If present, this will appear in a limited region over the cavity and usually be surrounded by dulness.

CRACK-POT RESONANCE. This may be heard if, while percussing over the cavity, one listens with his stethoscope, holding the receiver close to the patient's open mouth

RALES. Consonating and sometimes definitely metallic in quality, these will be heard unless the cavity is totally empty or filled with secretion.

Far-Advanced Disease

When this stage is reached, pronounced constitutional and focal symptoms are likely fever with night sweats, anorexia, loss of weight, productive cough, hoarseness, and complaints referable to other parts. However, if the proliferative response is decidedly predominant, the course may be so chronic that symp-



FIG 264 Far advanced bilateral caseous tuberculosis with cavitation in both upper lobes. (Courtesy Middlesex County Sanatorium, Waltham, Mass)

toms are surprisingly few. In the terminal stages, one may find the typical phthisical picture: weakness, emaciation, pallor, except for cheeks, which are sunken and likely to have the so-called hectic flush. Fingers may be clubbed, but this is usually a reflection of associated bronchiectasis.

Because of the infinite variety of changes which extensive tuberculous disease can produce in the lungs and the interaction of one change upon another, no particular combination of local signs can be said to be typical. A cavity, for example, may be obscured by surrounding solidification, an area of solidification by intrapleural fluid or air, or a diseased segment by surrounding normal lung or complementary emphysema Usually, however, one will find the following variants over one or more areas:

Restriction of Respiratory Motion. This may be uni- or bilateral, confined to one or both apices, or be widespread.

Solidification. This is more likely to be in the upper lobes.

Rales. Heard over any diseased region, these may be moist, consonating, fine, medium or coarse, depending on the nature of the process creating them

Cavitation. Advanced phthisis rarely occurs without cavitation.

Complementary Emphysema. This will be found in non-diseased regions.

Pleural Changes. Indications of thickened pleura, pleural effusion, pneumothorax or hydropneumothorax may be present.

Displacement of Intrathoracic Structures. Contraction of scar tissue, air, or fluid in a pleural cavity, or obstructive atelectasis may displace heart and mediastinal contents to one side or the other.

Tracheobronchial Local Tuberculosis

Tuberculosis of the wall of the trachea or a bronchus or both is a form of the disease not infrequently encountered in adults and usually associated with some degree of parenchymal involvement. In the early phase, the process is one of congestion and edema but it may progress to granulation, ulceration, or fibrosis with resultant moderate or pronounced stenosis of the airway Complete obstruction is possible. Localized wheezing and emphysema, or atelectasis of a segment may be evident This variant may be readily confused with bronchogenic tumor or obstruction from other cause. Bronchoscopic examination is imperative.

Tuberculoma

Occasionally tuberculosis will be reflected as a dense fairly circumscribed lesion discovered by x-ray, and representing a caseating mass with a fibrous capsule or a filled cavity. Communication with a bronchus may exist, thus making the lesion a potential source of bronchial dissemination Often there is no other evidence of disease, although sometimes a zone of tuberculous infiltration is present nearby or at a distance. Especially when it is the sole lesion, this process cannot be distinguished from tumor; presence of calcium deposits favors tuberculoma The uncertainty demands surgical intervention. Even in the older

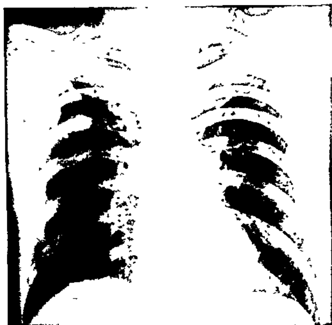


FIG 26 5 Tuberculoma Circumscribed, rounded, dense lesion, right upper lung field, discovered on routine film No symptoms or physical signs X-ray diagnosis Tuberculoma or tumor Diagnosis established following surgical removal of lesion

age group where the odds would appear to favor carcinoma, a fair proportion of cases turn out to be circumscribed tuberculosis

Chronic Fibroid Tuberculosis

Here the proliferative response predominates over the exudative so that fibrosis is more prominent than caseation and necrosis The process advances slowly, often covering 10 to 20 years or more. During this interval, general health is little impaired, focal symptoms are inconsequential, and, unless for some reason an x-ray is taken, the disease may exist for years without detection As the later stages are reached, the patient may complain of progressive dyspnea and productive cough; the latter results from associated bronchiectasis Recurrent hemoptysis may occur Tubercle bacilli may or may not be recovered from sputum or gastric secretion.

In the early phase, physical and x-ray signs may not differ from those of caseous tuberculosis, but as time goes on and parenchyma is replaced by fibrosis, a picture similar to that of chronic fibrosing pneumonitis develops. The thorax, uni- or bilaterally, is contracted and its expansion diminished, when extensive, contractures lead to thoracic asymmetry, deformity, and displacement of intrathoracic structures, usually toward the more badly diseased side. The pulmonary signs are variable The percussion note is mostly dull but areas



FIG 26 6 Chronic fibroid tuberculosis Extensive disease throughout both lungs, most marked in upper lobes Hilar shadows displaced upward, trachea toward right Heart shadow distorted Thickened pleura evident along right costal margin and in costophrenic angle Compensatory emphysema mid- and lower left lung fields (Courtesy Middlesex County Sanatorium, Waltham, Mass)

of hyperresonance due to complementary emphysema are usually found Breath and voice sounds may be diminished, bronchial, or bronchovesicular, and, in regions with cavitation, perhaps amphoric Rales, chiefly attributable to bronchiectasis, are the rule

Especially in communities where x-ray is not freely employed, fibroid tuberculosis, because of its chronicity and indolence, is particularly dangerous from the standpoint of contagion.

DIAGNOSIS

The diagnosis of pulmonary tuberculosis in its early stage is often extremely difficult. This is one of the serious drawbacks in the campaign against the disease since the chances of effecting a cure as well as preventing infection of others are much greater with early recognition. Every means of examination must be exhausted in the suspicious case. The patient's past must be carefully investigated for possible exposure and for any previous illness which might have been on a tuberculous background Symptoms such as weakness, fatigue, malaise, feverishness, loss of weight, irritability, and indigestion are always to be regarded with suspicion. So are persistent cough, frequent "colds," "bronchitis," or "virus

infection," hemoptysis, pleurisy with effusion, persistently enlarged lymphnodes, and ischeorectal abscess or fistula. Pleural effusion or hemoptysis, although often due to some other cause, should be regarded as tuberculous *until proved otherwise.* Any of the physical signs discussed in the preceding paragraphs are suggestive, especially rales, or indications of infiltration in the upper lung fields *That pulmonary tuberculosis, even when far advanced, can exist without symptoms or physical signs must always be kept in mind.*

Today positive diagnosis depends largely on x-ray, and bacteriologic isola- [illegible] This statement is not intended to discount the value of

[illegible]

tion. X-ray alone can be misleading, as, for example, in sarcoidosis or fungus infection, bronchiectasis, fibrosing pneumonitis, and cases showing local atelectasis or tuberculoma, which can so readily be confused with tumor.

No matter how suggestive the x-ray and clinical findings, every effort should be made to confirm or exclude tuberculosis bacteriologically. In the ascending order of effectiveness and reliability, these are: direct smear, smear of concentrated material, culture, and guinea pig inoculation Because, especially in the early case, the organism may be elusive, repeated examinations are often imperative, one or two negative tests by no means exclude the disease. Depending on circumstances, sputum, gastric secretion, pleural effusion, and/or material

[illegible]

case the existence of tuberculosis elsewhere can be proved, as for example, by [illegible] ine, or [illegible], bron- [illegible] Papanicolaou method. Sometimes the diagnosis can be established pathologically by biopsy of a lymphnode or other diseased part, in the case of a circumscribed pulmonary lesion, surgical exploration is often indicated.

The tuberculin test may be helpful A negative test excludes tuberculosis except in overwhelming disease or in the rare group of cases which show a negative response in the presence of old calcified lesions A positive test indicates tuberculous infection A decade or more ago it was likely to reflect in the adult only sensitization due to a long-healed primary infection; now that childhood disease is less common in this country, the positive test in the adult carries greater weight and can be regarded with more assurance as an indication of a more recent or active infection

Status

Recommendations of the National Tuberculosis Association for indicating the status of the patient with pulmonary tuberculosis are summarized as follows:[2]

[2] Ibid

Inactive. Lesions observed in serial x-rays stable except for extremely slow shrinkage No evidence of cavity.

Symptoms absent

Sputum, if any, repeatedly negative, not only by concentration and microscopic examination, but also by culture or animal *inoculation* When adequate sputum not available or the findings doubtful, studies of gastric secretion or secretion aspirated from tracheobronchial tree must be negative by culture or animal inoculation

These conditions shall have existed for at least 6 months The period of inactivity shall be designated, if known It does not include and is in addition to the 6 months required for determination of inactivity

Active. Lesions observed in serial x-rays usually progressive or retrogressive, but may be stationary

Symptoms usually present, but may be absent

Sputum and gastric contents almost always contain tubercle bacilli although, in some instances, tubercle bacilli cannot be demonstrated even after repeated cultures and animal inoculations

Tuberculin test almost always positive

Pulmonary lesion will be classified as active whenever an empyema, bronchopleural or pleurocutaneous fistula, or active endobronchial tuberculosis is present

The period of activity shall be designated, if known The designation *Active, Improved* or *Active, Unimproved* may be used after an adequate period of observation or treatment

Activity Undetermined. When activity has not been determined from adequate x ray and laboratory examinations, the disease may be designated temporarily as *Activity Undetermined* If a provisional estimate of the probable clinical status is necessary for public health purposes, the term *Probably Active* or *Probably Inactive* should be used

In any of the above groups, if the patient is receiving chemotherapy, the word *Chemotherapy* should be added in parenthesis to the classification, it should not be used if the treatment has been terminated prior to the time of classification

The Association also recommends that the exercise status of the patient be indicated, as bedrest, semi-ambulatory, ambulatory, returned to ordinary living conditions The details of this classification are not pertinent to this book

ACUTE TUBERCULOUS PNEUMONIA

This term is applied to cases in which profuse bronchial dissemination of organisms creates an extensive acute exudative reaction with severe systemic response

Tuberculous Lobar Pneumonia

Relatively rare and appearing chiefly in adults, this arises from an already existing focus but almost always in a person who is thought to be in good health During the first week or so, it often cannot be distinguished from pneumococcus lobar pneumonia Onset is abrupt, with high fever, prostration, and perhaps a chill. Dyspnea and productive or non-productive cough appear early. Sputum, if present, may be blood-tinged or have the typical rusty appearance characteristic of pneumococcus infection At this stage, tubercle bacilli are not detectable. By x-ray and physical examination one finds indications of solidification indistinguishable from those of pneumococcus lobar pneumonia; part or all of a lobe or an entire lung may be involved.



FIG 26 7. Tuberculous lobar pneumonia, right upper lobe Some diffuse infiltration in right mid-lung field is also evident (Courtesy Middlesex County Sanatorium, Waltham, Mass)

If specific therapy is not initiated, the clinical picture changes after the first or second week Temperature takes wider swings; cough, dyspnea, and weakness increase, pulmonary softening and cavitation are indicated by increasing number of moist rales and expectoration of larger amounts of mucopurulent sputum, in which tubercle baccilli can almost always be found Progression downward is usually rapid, the patient dying within 6 or 8 weeks.

Prompt and adequate specific therapy will result, after a few months, in pronounced improvement, but as a rule, resolution is not complete, an area of density, sometimes with a cavity, may persist

Especially in its early phase, the disease so closely resembles pneumococcus pneumonia that differentiation between the two is virtually impossible The problem is further complicated by the fact that pneumococcus lobar pneumonia can appear in a person known to have pulmonary tuberculosis Bacteriologic studies and the response to antibiotic therapy will usually eventually provide the answer Tuberculosis must be kept in mind in any case initially thought to be pneumococcus lobar pneumonia which does not promptly improve with specific therapy

TUBERCULOUS BRONCHOPNEUMONIA

More common than tuberculous lobar pneumonia, this is usually encountered in persons with known tuberculosis Often it follows an attack of upper respiratory infection, some infectious disease such as measles or whooping cough, anesthesia, prolonged fatigue or exposure, or malnutrition It has a preference for children and young adults

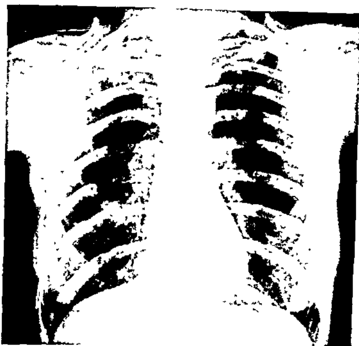


FIG 26 8 Tuberculous bronchopneumonia Diffuse infiltration with patchy areas of solidification throughout right lung field (Courtesy Cambridge Sanatorium, Cambridge, Mass)

Onset is usually gradual with fever, and loss of appetite and strength. The temperature chart shows wide daily swings Sometimes hemoptysis is the first symptom. Recurrent chills and night sweats, loss of weight, dyspnea, cyanosis, and productive cough are the rule

Usually in the upper lobes, the local signs initially are similar to those of early or moderately advanced caseous tuberculosis, sometimes the picture resembles that of acute bronchitis Signs of solidification appear in short order, at first in small scattered patches in one or more lobes but soon coalescing to form larger areas Rales of various types are heard Caseation and cavitation develop within a short time, tubercle bacilli are found in the sputum early in the course Diagnosis as a rule is not difficult, especially if one keeps in mind that tuberculosis can produce a clinical picture similar to that of ordinary bronchopneumonia

With the use of specific therapy, the prognosis is much more favorable than heretofore Many cases will clear entirely although a few may be left with a low-grade progressive lesion.

MILIARY TUBERCULOSIS

ACUTE

In this form of tuberculosis, minute tubercles are diffusely distributed throughout the body by blood stream dissemination of organisms from a caseous pulmonary or lymphnode lesion following erosion into a vessel. Occasionally a focus in some other structure, such as a kidney or bone, may be the source. Although

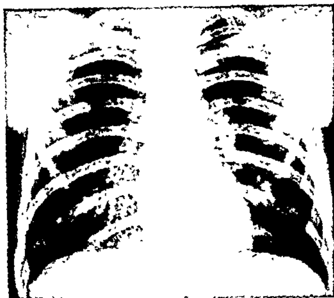


FIG 269 Miliary pulmonary tuberculosis Fine mottling throughout, most prominent in right ower lung field (Courtesy Middlesex County Sanatorium, Waltham, Mass)

dissemination is widespread, certain structures—the thyroid gland, skeletal muscles, brain, and stomach—are rarely appreciably affected even while others show extensive disease

Onset may be sudden with high fever and prostration, or insidious with gradual development of fever, malaise and weakness Temperature averages 102°–104°; the chart is often irregular with pronounced swings Because the pulmonary lesions are interstitial, cough and expectoration are not striking except in the late stages, tubercle bacilli are discoverable in the sputum only when the process has reached the point of parenchymal breakdown Dyspnea, cyanosis, and increased respiratory rate are often prominent and out of proportion to physical signs of trouble in the lungs During the early phase, x-rays of lungs are not helpful except that, by showing evidence of a local pulmonary or lymphnode lesion, they might lend support to one's suspicion that an unexplained febrile illness is miliary tuberculosis. When the miliary lesions become large enough to be visible one will see diffuse, small, homogeneous densities scattered throughout both fields The picture cannot be roentgenographically distinguished from other diffuse pulmonary diseases such as sarcoidosis and mycotic infection

As noted above, sputum is negative at the start, it may remain so for weeks Seeding elsewhere, as, for example, in the meninges, pleura, or peritoneum, may create an identifiable process Early diagnosis is sometimes possible by culture, animal inoculation or microscopic study of bone marrow

Miliary tuberculosis must be suspected in any case of unexplained fever of more than a few days' duration, especially if there is evidence of a recent or old

tuberculous focus anywhere in the body, but failure to demonstrate it by no means excludes the disease. It must be distinguished from other maladies causing prolonged fever, such as lymphoma, carcinomatosis, mycotic infection, subacute bacterial endocarditis, brucellosis, and typhoid fever.

Formerly this form of the disease had a very high mortality rate. Cases with meningitis were almost invariably fatal. In recent years, specific therapy, especially when started promptly, has made the prognosis less grave, although in cases developing meningitis, death or total incapacity remains a likely outcome.

Subacute

Sometimes a less overwhelming dissemination of tubercle bacilli will create a less acute disease in one or more structures. Pulmonary lesions have already been described. Others are lymphnodopathy, pleuritis, peritonitis, pericarditis, or disease of bone, eyes or parts of the genito-urinary system They may cause trouble or become entirely healed without creating signs or symptoms.

REFERENCE

1 *Diagnostic Standards and Classification of Tuberculosis* New York, National Tuberculosis Association, 1955

MISCELLANEOUS DISEASES OF THE LUNGS

ABSCESS

Lung abscess is a local area of parenchymal suppuration, usually producing a cavity which may vary from a few mm to 10–12 cm. in diameter. One of the following causes is most likely.

1. Operation on the mouth or upper respiratory tract, especially tonsillectomy or tooth extraction. This accounts for a high proportion of cases
2. Bronchial obstruction, especially tumor or foreign body.
3. Abolition of cough reflex, as in prolonged unconsciousness or anesthesia, with resultant inspiration of infected material
4. Necrosis within a pulmonary infarct

Abscess occasionally follows lobar pneumonia or results from extension of infection from an adjacent region such as a subdiaphragmatic abscess. Multiple scattered abscesses are most likely due to septic emboli from a focus elsewhere

Symptoms

In the early stage of development these depend on the cause. Following an operation, especially in one of the regions noted above, abscess should be suspected if cough, pleural pain, expectoration, fever, and perhaps chills develop, although the possibility of infarction or pneumonia should not be overlooked. Abscess following inhalation of a foreign body has a similar onset but again it must be stressed that in a child, no history of the episode may be obtainable In postoperative and inhalation cases the constitutional symptoms are usually pronounced high fever, usually septic in type, sweats, chills, and marked prostration *In abscess following slowly developing bronchial obstruction* such as tumor, onset is more gradual and systemic response at the start usually less severe. Following pneumonia, abscess is suggested by persistence of symptoms, especially if, after 10 or 12 days from onset, systemic reaction becomes intensified, the chart becomes septic in character, recurrent chills appear, or cough becomes increased

Sudden expectoration of a large amount of foul, purulent, sometimes bloody sputum, after which the patient may experience a sense of relief, is an important indication of abscess. It is due to rupture of the lesion into a bronchus.

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Sudden expectoration of a large amount of foul, purulent, sometimes bloody sputum, after which the patient may experience a sense of relief, is an important indication of abscess It is due to rupture of the lesion into a bronchus.

Subsequently, periodic attacks of cough productive of the same type of sputum occur they are often precipitated by shift of the patient to a particular position which favors drainage. The breath is extremely foul

In general sepsis, productive cough and pleural pain suggest embolic abscess formation, other symptoms are masked by those of the septic state itself.

Signs

Pulmonary Infiltration. The local signs of abscess are influenced by such factors as the age of the lesion, type and amount of surrounding parenchymal inflammation, presence of a cavity, its size, and the amount of material it contains Usually the picture is a reflection of infiltration of surrounding lung parenchyma· dulness, bronchial breathing, and bronchial whispered and spoken voice sounds Friction rub and crepitant rales may be heard Sometimes, especially if the lesion is small or deep-seated, overlying normal lung may entirely mask those which can be attributed to the abscess, or the latter may not be evident because of changes due to the primary disease, as for example, in total obstruction of a bronchus due to tumor where no air is going into the affected region Embolic abscesses show no characteristic signs There are usually a few scattered rales and there may be small areas of pleural friction or, occasionally, of slightly altered resonance or breath sounds Embolic abscesses are apt to be associated with purulent pleuritis and masked by its signs. Long-standing chronic abscess may show only the indications of local pulmonary fibrosis

Cavitation. The distinctive signs of an abscess cavity are often absent because of surrounding inflammatory reaction or bronchial obstruction However, if the cavity is fairly large and not filled with exudate, one will find

1 Tympany

2 Amphoric breath sounds.

3 Bronchial whispered voice sounds Sometimes they have an amphoric quality

4 Consonating rales. These have an almost metallic quality They are not heard if the cavity is totally empty

Clubbing of Digits. This is common in lung abscess and likely to become pronounced within a few weeks of onset. Other indications of pulmonary osteoarthropathy may appear

X-Ray Findings

Initially the picture is indistinguishable from any local inflammatory process, later one sees an irregular rounded density with a central rarefaction which may or may not show a fluid level In contrast to tumor, the wall is rarely nodular but may be shaggy and irregular.

Sputum

Once an abscess has ruptured, sputum is very profuse, often amounting to as much as 300–500 cc. within 24 hours It is purulent, very foul, sometimes blood-tinged. Frank hemorrhage may occur and is sometimes fatal. On macroscopic

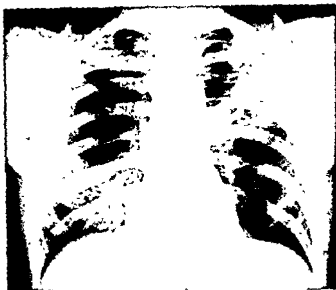


FIG 27 1 Abscess cavity in a suppurative process, left upper lobe, resulting from inhalation of vomitus Right lower lobe shows similar infiltration but no cavitation Both lesions cleared completely following appropriate antibiotic therapy (Courtesy Cambridge Sanatorium, Cambridge, Mass)

examination one may find small particles identified as necrotic pulmonary tissue, which, if teased out on a slide and examined microscopically, will show an alveolar pattern—evidence that they come from the lung Elastic fibers may be seen. Also visible microscopically are large numbers of leukocytes in various stages of degeneration and numerous bacteria of different types, particularly diphtheroids, streptococci, fusiform bacilli, spirochetes and fungi. In abscess following infarction, blood is usually present in the early stages, and later hematoidin crystals may be found.

Diagnostic Pitfalls

The possibility of lung abscess must always be considered in any case showing evidence of some pulmonary disorder developing after operation, especially in the mouth, nose, or throat, or under any of the other circumstances noted above as favoring abscess formation *Bronchoscopic examination is often essential.*

Tuberculous Cavitation The course of tuberculosis is more chronic and accompanied by more widespread evidence of pulmonary involvement, demonstrable by physical and x-ray examination Demonstration of tubercle bacilli in the sputum or gastric contents will establish diagnosis

Bronchiectasis. This is also more chronic and gives rise to fewer constitutional symptoms Examination of the sputum is helpful but diagnosis is best established by x-ray studies, including bronchograms, and by bronchoscopy.

Empyema with Rupture into a Bronchus. This usually follows pneumonia and, like abscess, may first be suspected when there is sudden expectoration of

a large amount of purulent sputum followed by productive paroxysmal cough. Physical, x-ray and bronchoscopic studies will usually establish diagnosis. Microscopically the sputum in ruptured empyema shows only one type of organism, usually pneumococcus, while that of pulmonary abscess shows *mixed flora.*

GANGRENE OF LUNG

This rare malady cannot be distinguished clinically from lung abscess It is usually a *post-mortem* finding. one or more large areas of necrotic pulmonary tissue, greenish-black in color, foul and interspersed with areas of cavitation containing foul fluid.

It is invariably secondary to some underlying pulmonary disease, commonly bronchiectasis, pulmonary embolism, bronchial obstruction, septic bronchopneumonia and, rarely, lobar pneumonia

Symptoms are very similar to those of lung abscess Systemic reaction is severe and death almost always the end-result Physical signs are variable. In the early stages there may be evidence of pulmonary consolidation, later, signs of cavitation. The most outstanding feature is the sputum which is profuse, greenish-black and nauseatingly offensive Microscopic examination shows elastic and, sometimes, large fragments of pulmonary tissue.

X-ray will show extensive mottled infiltration with interspersed patches of solidification and often rarefied areas representing multiple small abscesses. *Associated pleural* effusion may conceal the parenchymal changes.

PULMONARY EMBOLISM AND INFARCTION

Pulmonary *embolism* is the lodging of foreign material in the pulmonary artery or one of its branches with resultant impairment of blood flow In the lung because of its double blood supply embolism does not necessarily predicate infarction.

Pulmonary *infarction* is necrosis of parenchyma secondary to arrested circulation. This is most likely to occur when the embolus is large or infected, or when the lung is otherwise impaired, as by atelectasis following operation or edema secondary to congestive failure It is least likely when the embolus is small and not infected, and the lung normal.

Pulmonary emboli usually arise from

1. Thrombophlebitis or phlebothrombosis (*see* Chap 34). Occurring in the lower extremities, pelvis, or abdomen, either is most likely encountered following operation, after direct injury to an extremity, in severe infectious disease, congestive heart failure, carcinoma, especially of the pancreas, and under other circumstances, such as long immobilization, which favor venous stasis As a rule, emboli due to these causes are not infected.

2. Thrombus in the right auricle in long-standing heart disease, especially mitral stenosis, or, less commonly, a mural thrombus in the right ventricle due to myocardial infarction which has extended to the right side of the heart.

3. Valvular vegetation in a right heart chamber in acute or subacute bacterial endocarditis.

Emboli arising from a valvular vegetation or from an infected vein in a septic area, as in pylephlebitis secondary to acute appendicitis with rupture, are almost always multiple, infected, and usually give rise to multiple small abscess formations in the lungs, occasionally to a single abscess.

Non-Septic Embolism

Small Embolus

An embolus obstructing a small or medium-sized artery may not produce an infarct sufficiently large to cause any appreciable mischief. It is possible, however, that the transient attack of faintness sometimes encountered during the week or so following operation, or under other circumstances favoring phlebothrombosis, may be a manifestation of this phenomenon. Some authorities believe that, in a suspected case, enthusiastic examination, especially auscultation, is inadvisable because of the possibility that moving the patient about or increasing negative pressure in the thorax by deep inspiration may favor dislodgement of another clot from the impaired vein

If clinical indications of a relatively small infarction are present one will usually find one or more of the following variants, but, except for pain, they may not be evident for two or three days

Localized Pain in Thorax. Due to regional pleuritis, this appears suddenly, is sharp, increased by respiration, and lasts for only a few hours or days.

Cough. This is dry or productive of blood-tinged or frankly bloody sputum.

Elevation of Temperature, Pulse and Respiration.

Local Friction Rub. Its absence by no means excludes infarction



Fig 27 2 Pulmonary infarcts in a patient with thrombophlebitis of lower extremities Film taken 10 days after onset of dyspnea and right pleuritic pain. Sharply outlined area of density at periphery of right mid-lung field and another at base

Signs of Solidification. A small area of dulness, bronchial breathing and whisper may appear a day or so after onset and last for a few days but often it is not detectable.

X-ray Findings. No changes appear unless infarction occurs. When it does the films may be negative for a couple of days or longer Later a sharply defined small area of density will appear in contact with a peripheral or interlobar pleural surface. Its shape is usually pyramidal with its widest base on the periphery and a somewhat rounded apex pointing toward the hilus Since infarct can best be identified when seen in profile, several projections are often essential.

Moderate-Sized Embolus

When an embolus obstructs one of the larger branches of the pulmonary artery, onset is marked by.

Sense of Suffocation. This appears abruptly Sometimes the patient complains more of pain, tightness, or oppression in the chest Dyspnea is obvious

Distended Cervical Veins.

Cyanosis.

Accentuation of Pulmonic Second Sound.

Signs of Peripheral Circulatory Collapse.

Indications of Infarction. If, as usually happens, the patient survives, the above indications of the acute insult will disappear within a day or so and be followed by cough, bloody sputum, fever, signs of solidification in all or part of a lobe and, usually, pleural pain and friction It must be emphasized, however, that these signs may be lacking.

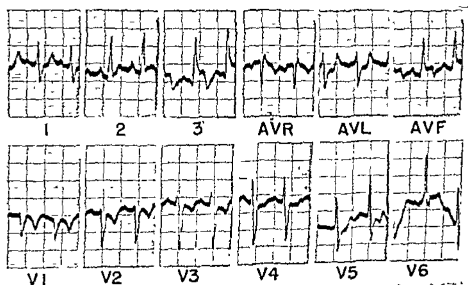


Fig 27 3 Pulmonary embolism Tracing taken ½ hour following sudden onset of dyspnea in a patient with thrombophlebitis of lower extremities Death occurred ½ hour later S wave in Lead 1 Q wave in Lead 3 T waves inverted in Leads 3, AVF, and V1-V3

X-ray Findings. The picture is similar to that described above except that the lesion is larger [illegible]

[illegible] tion, dissecting aneurysm, massive collapse, and pneumonia If changes appear one will find prominent S1 and Q3, and T wave inversion in leads 2, 3, AVF and the right-sided precordial leads (V1–V3). These variants are thought to represent acute right heart strain but may, to some extent, reflect sudden rotation of the heart on its long axis.

Following operation, in mitral stenosis especially with auricular fibrillation, congestive failure, and other circumstances favoring venous stasis, the appearance of unexplained fever, tachycardia, faintness, dyspnea, new signs in the lungs, or an episode resembling coronary insufficiency or myocardial infarction, is highly suggestive of pulmonary embolism of this degree The lesion, especially when postoperative, is frequently confused with pneumonia or massive collapse Infarction usually appears later than either of the others Other important differences are abruptness of onset and, in some cases, the physical findings and distinctive x-ray and electrocardiographic changes

Large Embolus

Massive embolism involving the pulmonary artery or either of its primary branches is a fairly frequent cause of sudden death, particularly after operation, and, sometimes, in heart disease, especially predominating mitral stenosis with fibrillation or congestive failure from any cause Multiple emboli resulting from fragmentation of a large one and obstructing a number of smaller arteries may act in a similar way and be clinically indistinguishable from a single large embolus The postoperative cases usually occur from 1–2 weeks following operation, the existence of venous thrombosis is often unsuspected beforehand. A frequent occurrence, especially in the postoperative case, is that just before the catastrophe, the patient calls for a bedpan It may be that exertion attendant upon the attempt to defecate causes dislodgement of the clot The premise has also been advanced that, on the contrary, the sensation of needing to defecate is created by breaking off of an intra-abdominal clot. The outstanding features of large embolus are

Substernal Oppression The patient suddenly complains of a feeling of suffocation, inability to get the breath, or severe tightness across the chest Dyspnea is extreme

Cyanosis.

Distended Cervical Veins.

Accentuation of Pulmonic Second Sound.

Collapse.

Death is likely within minutes to hours If the patient survives, signs of infarction involving a small or large segment of lung or, sometimes, pleural effu-

sion may develop within a few days. Often, however, canalization in the affected vessels and collateral circulation from the bronchial arteries will prevent formation of an infarct. In some cases impaired circulation, due to the clot in the major vessel and thrombosis extending distally, will eventually cause chronic cor pulmonale

X-Ray Findings. If one of the major pulmonary arteries is occluded without creating infarction, the shadow of the proximal pulmonary artery appears increased in size, and one notes a sharp change in caliber of the vessel at the point where the embolus lodges. Distally, the arteries are smaller than their counterparts in the unaffected lung If infarction occurs, a large homogeneous area of density shaped like a rounded pyramid and with its base on a plural surface will be seen. Multiple small infarctions secondary to a fragmented large embolus may be observed, but this is uncommon.

Electrocardiographic Findings. (*See above*)

Negative x-rays and absence of abnormal physical signs in the lungs, especially within the first few days after onset of an episode, by no means exclude pulmonary embolism or infarction

Septic Embolism

When infected emboli reach the lung, septic infarcts are formed Septic bronchopneumonia or multiple small pulmonary abscesses may develop, associated purulent pleuritis is not uncommon The indications of pulmonary involvement are often obscured by those of the overwhelming septicemia Cough, *pleural pain, purulent sputum, rales, or signs of small patches of solidification* in the lungs or of pleural effusion, occurring in a septic case, should alert one to the possibility of embolic disease Occasionally a single infected embolus will cause isolated septic infarct which will develop into an abscess

Fat Embolism

In a considerable number of patients dying after major trauma with or without fracture, fat emboli are found in the smaller pulmonary arteries, arterioles, or alveolar capillaries. Cough, dyspnea, fever, cyanosis, and physical and x-ray findings simulating bronchopneumonia or pulmonary edema may have been present, usually starting several days *following the injury* Authorities disagree as to whether pulmonary fat embolism can actually be responsible for death; although found in some traumatic cases with terminal respiratory symptoms, it is totally absent in others In the pertinent case, fat globules in the urine are highly suggestive; in the sputum they are of less significance. Occasionally, autopsy will show that fat, presumably after traversing the pulmonary circuit, has terminated in kidneys or brain (*see* Chap 37)

ATELECTASIS

Functional Atelectasis

During ordinary respiration the normal person does not completely inflate his lungs. If their margins at the bases and in lower axillae are *identified by per-*

cussion and auscultation, first during quiet breathing and then following deep breathing, it will be noted that the latter has extended the borders beyond the former limits This change indicates a slight degree of marginal atelectasis. A few fine rales (*atelectatic crepitation*) may be heard in these regions during the forced inspiration, especially in older persons or those who have been inactive for a long time.

In *atelectasis of the newborn*, which is usually fatal within a few hours to 1-2 days, the lungs are anatomically normal but do not properly expand because of impairment of respiratory movement due to some disturbance, usually anoxia, in the brain stem

Obstructive Atelectasis

All or part of a lung is deflated because of impaired inflow due to obstruction of its airway by exudate, foreign body, tumor, or stenosis

Symptoms

As a rule, symptoms attributable to the atelectasis itself occur only when a large area is involved and usually when obstruction appears so suddenly that the pulmonary system does not have time to adjust to the ventilatory impairment In this event, dyspnea will occur Otherwise what symptoms are present are related to the underlying disturbance

Signs

Because the bronchus is closed and no air is entering the affected segment, one will find

Dulness to Flatness.



FIG 27 4 Obstructive atelectasis Total collapse of left lung due to obstruction of primary bronchus by adenoma Trachea, heart and mediastinal structures displaced to left.

Absent Tactile Fremitus.

Absent Breath, Whisper, and Voice Sounds.

Absence of Rales.

Displacement of Adjacent Structures. If a large segment of lung is involved, one may observe displacement of heart and mediastinal contents toward the diseased side, homolateral upward displacement of diaphragm, and narrowing of intercostal spaces. These changes are helpful in distinguishing obstructive atelectasis from pleural effusion, pneumonia, and infarction

X-Ray Findings

If a bronchus is obstructed one will see reduction in size with somewhat increased density of the involved segment or lobe Evidence of inflammatory change distal to the obstruction will become evident if the cause is not soon removed Displacement of heart and mediastinal structures, elevation of diaphragm, and narrowing of intercostal spaces may be apparent in obstruction of a major bronchus

Postoperative Atelectasis

Although this is a form of obstructive atelectasis due to plugging of a bronchus by exudate, it deserves special mention The patient develops dyspnea and cough, temperature, pulse, and respiratory rates are elevated, and the systemic reaction is often severe At onset, one finds dulness to flatness, absent tactile fremitus, breath, and voice sounds, and no rales over a fairly large area But

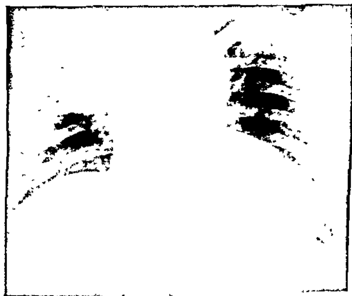


Fig 27 5 Postoperative atelectasis Film taken 5 days after intra abdominal operation Dense shadow of collapsed right upper lobe and homolateral elevation of diaphragm Complete clearing within 24 hours after bronchoscopic aspiration of mucoid secretion obstructing right upper lobe bronchus

within a short time the signs change to those of solidification whereupon confusion with postoperative pneumonia or infarction is likely. The differential diagnosis depends on the demonstration of elevation of the diaphragm and displacement of heart and mediastinal contents toward the affected side This can sometimes be detected by physical examination, at other times only by x-ray. The latter will sometimes show, in addition, downward inclination of the ribs and narrowing of the intercostal spaces on the homolateral side.

Change of position or sudden cough often dislodges the bronchial exudate and the atelectasis disappears suddenly or gradually In a doubtful or serious case, bronchoscopy is indicated, not only for diagnosis, but also because, if collapse due to retained secretion is the cause of the trouble, the patient's condition can be greatly improved by suction.

Compressive Atelectasis

All or part of the lung is compressed by fluid or air in the pleural cavity, pericardial effusion, greatly enlarged heart, large intrathoracic tumor, or elevation of the diaphragm from paralysis or pressure from below. Small areas of atelectasis are often present with various pathologic states such as tuberculosis, pneumonia, and chronic bronchopulmonary disease due to various causes They do not create any characteristic clinical signs

Symptoms

If a large area of lung *suddenly* becomes atelectatic as in spontaneous pneumothorax, dyspnea, and perhaps circulatory collapse will appear If compression occurs *gradually*, as from slow accumulation of pleural fluid, symptoms are less pronounced or absent.

Signs

A small atelectatic segment will not be apparent on physical examination Over a large area, depending on whether the bronchus is open or closed, one will find the typical signs of solidification, or flatness, and absence of sounds which reflect the total impairment of local ventilation. In the case of atelectasis due to fluid or air, the diaphragm will be displaced downward and the mediastinal contents away from the affected area, as opposed to obstructive atelectasis, in which they are displaced toward the lesion

X-Ray Findings

The picture is that of the underlying process with reduction *in size* and *increased* density of the affected portions of the lungs.

PULMONARY EDEMA

Acute

A secondary process, this is seen in acute cardiac dilatation, during the course of chronic heart disease especially left ventricular strain or predominating mi-

tral stenosis, and sometimes in renal failure, the terminal stage of a severe, acute infection, or a comatose state. It may also be induced by improper regulation of electrolyte balance or overloading of the circulation with blood, blood substitute or fluid.

The attack starts suddenly with extreme dyspnea, a feeling of oppression in the chest, constant cough, and quite often cyanosis Large quantities of pinkish, frothy sputum are coughed up, virtually pouring from the mouth in the severe case The pulmonary signs are rhonchi, coarse or medium moist, and occasionally, sibilant rales They are so numerous that breath and heart sounds are obscured

Chronic

This too is most common in left ventricular failure and predominating mitral stenosis, but is also found in other disturbances marked by abnormal fluid retention and in debilitated long-bedridden patients In a mild case the only sign is medium or fine moist rales heard at the bases, or, when the patient has been lying mostly on one side, chiefly in the dependent lung In the long-standing case, dulness, diminished sounds, sometimes bronchovesicular, and diminished tactile and vocal fremitus are likely When fluid retention is marked, coexistent hydrothorax may obscure the pulmonary signs. Chronic pulmonary edema is distinguished from other disorders causing similar signs by absence of systemic reactions such as fever, and demonstration of a cause for its existence

In acute or chronic edema, x-ray shows hazy, homogeneous density in the



Fig 27 6. Pulmonary edema in a case of rheumatic heart disease with predominant aortic stenosis Typical "butterfly" distribution of hazy, homogeneous densities with ill-defined margins, extending from the hili into mid-lung fields. Apices and bases not involved

central lung fields forming the so-called *butterfly pattern*. If the heart is at fault, its shadow may reflect the underlying disturbance. Occasionally edema is limited to one lung or even to a single lobe.

TUMOR OF LUNG

Primary tumor of lung is most likely bronchogenic. It may occur as a single circumscribed mass or in an infiltrative form. Occasionally it is marked by multiple nodules, one of which is the primary *focus, the others, metastases*. Metastatic involvement of the opposite lung is not common. Lymphoma may appear in a multiple nodular or infiltrative form. Pulmonary adenomatosis, a rare primary tumor, shows progressive diffuse infiltration and, *due to involvement* of alveoli, areas of solidification. Many observers believe that adenomatosis and alveolar cell carcinoma are the same disease. In any case of suspected tumor, Papanicolaou study of sputum and of secretion obtained through the bronchoscope should be performed.

Secondary tumor occurs by extension or metastasis from disease elsewhere, the most common sources being, (1) carcinoma of breast, stomach, pancreas, thyroid, kidney, prostate, testicle, or uterus, and (2) sarcoma, especially of bone.

Metastatic tumor may consist of small or large, single or multiple nodules, or the lung may be diffusely infiltrated (*miliary malignant disease*). It is more likely than primary tumor to be bilateral and show multiple lesions.

Localized Primary Tumor

Symptoms

Since this form is most apt to be bronchogenic adenoma or carcinoma, the *early* manifestations reflect changes in the bronchus

Cough. Often the initial complaint is constant hacking cough, possibly productive of scanty, mucoid, blood-tinged or bloody sputum Hemoptysis, when present, tends to persist for days or weeks, in contrast to the more profuse and periodic episodes of tuberculosis, bronchiectasis, and other forms of pulmonary disease Frank hemorrhage is rare in carcinoma, occasional in adenoma. Because of the cough and perhaps some fever—attributable to secondary infection—early tumor is often incorrectly diagnosed virus pneumonia. *When a person who has enjoyed good health begins, unaccountably, to be nagged by a harassing cough, especially if it is accompanied by blood-tinged sputum, he should be very carefully studied for the presence of bronchial or lung tumor.*

Dyspnea. Occurring only when a large bronchus is obstructed, this is often paroxysmal and may be of the asthmatic type.

Pain. This is not an early symptom except in carcinoma involving the apex. Invasion of the first dorsal root of the brachial plexus causes pain down the arm and, if the second intercostal nerve is involved, along the inner aspect of the upper arm Extensive disease may cause pain by invading pleura, mediastinum or other intrathoracic structure.

In the *late* stages, the tumor itself or secondary infection will create such constitutional symptoms as fatigue, anorexia, feverishness, and loss of weight. Invasion of pleura, ribs, or intercostal nerves will cause thoracic pain, of the mediastinum, substernal or mid-back pain, and perhaps dysphagia, hoarseness or, due to high obstruction, severe dyspnea.

Signs

Progressive Bronchial Obstruction. When the tumor is bronchogenic, the indications of progressive obstruction appear first (*see* Chap 24) Expected displacement of *heart and other structures toward the affected side* may be absent because of fixation due to mediastinal invasion If a pulmonary apex is involved, Horner's syndrome may be observed.

Indications of Infection. Bronchiectasis, pneumonitis with or without pleuritis, or lung abscess is likely to develop when obstruction reaches the point of creating stagnation.

Signs of Pleural Effusion. (*See* Chap 28) Direct extension or lymphatic seeding into the pleura with resultant effusion is not uncommon. This may be the first indication of trouble. If an appreciable amount of fluid is present, it will obscure the physical and x-ray signs of pulmonary change Thoracentesis is indicated Bloody fluid is highly suggestive of malignant disease, diagnosis can often be established by Papanicolaou smear or cell block study of centrifuged specimens

Obstruction of a major bronchus, by causing obstructive atelectasis of the lung, may create signs simulating those of fluid, here, unless fixed by tumor, the heart and mediastinal structures will be pulled toward the affected side A large tumor extending to the periphery can cause flatness, absence of tactile fremitus, and absence or pronounced diminution of breath, whisper, and voice sounds On percussion, more resistance is felt than in any other pulmonary disease, the very flat percussion note and pronounced sense of resistance favor tumor as opposed to fluid. As a rule, these various possibilities can be differentiated by x-ray but thoracentesis is sometimes necessary

Mediastinal Obstruction. Venous distention, edema, cyanosis of the neck and upper extremities, and, sometimes, collateral dilatation of the superficial veins of the chest and abdomen appear if the mediastinum is sufficiently invaded to cause interference with return flow of blood

X-Ray Findings

Localized tumor, when peripheral, is usually homogeneous, rounded, somewhat nodular, or irregular in contour, and may or may not have a smooth, *clearly* defined edge Calcification is not uncommon in benign lesions such as hamartoma and tuberculoma but, with the exception of osteogenic sarcoma, is *rare in* malignant disease As a rule, bronchogenic carcinoma arises in a major bronchus close to the hilus Initially, the tumor may be seen in the bronchus as a *small,* circumscribed mass partly outlined by air or as a larger, often poorly outlined area of density projecting *from the hilus outward into the lung field.*



FIG 27 7 Bronchogenic carcinoma, lingular segment, left upper lobe Area of greatest density adjacent to heart border represents tumor mass The less dense shadow extending laterally is caused by collapse and inflammatory infiltration of pulmonary segment distal to point of bronchial obstruction

Peripherally, in the early stage, one may see a zone of obstructive emphysema involving a small or large segment of lung or even an entire lung, depending on the size of the partially occluded bronchus. Later, when complete obstruction has ensued, the picture will be that of atelectasis—a wedge-shaped area of density extending outward from hilus to periphery. In time this shadow will become irregular and mottled due to infiltration and bronchiectasis resulting from secondary infection due to impaired drainage At this stage it is often impossible, radiologically, to distinguish between tumor and atypical pneumonia or other infectious process

Localized Metastatic Tumor

One or more isolated, fairly circumscribed lesions may develop in the lungs and grow to considerable size without causing symptoms or signs. Often they are discovered by x-ray survey Or, like primary tumor, a metastatic lesion can produce the signs of bronchial obstruction, mediastinal invasion, or extensive involvement of lung parenchyma. Constitutional symptoms appear in the later stages X-ray changes are similar to those described above Often it is impossible radiologically to distinguish a primary from a metastatic lesion.

Diffuse Infiltrative Tumor

Rare in primary carcinoma, diffuse lymphatic spread through the lungs is not uncommon in metastatic disease and lymphoma Cough with or without slight



FIG 27 8 Metastatic tumor Rounded, sharply-defined, homogeneous shadow in right lung, Primary lesion Fibrosarcoma of hand



FIG 27 9 Multiple circumscribed tumors of lungs, metastatic from carcinoma of cervix Mediastinal shadow irregularly widened by metastatic lymphnode enlargement

hemoptysis may be the first symptom Dyspnea is at first mild but gradually becomes pronounced, much greater in fact than would be expected on the basis of the physical and x-ray findings Pain is a feature only with peripheral or mediastinal involvement Constitutional symptoms of extensive malignant disease are the rule. *Except when pleural involvement causes thickening or effusion,* local physical signs are usually negligible In spite of extensive disease and even in



FIG 27 10 Diffuse mottling throughout both lungs representing metastatic spread from carcinoma of stomach Hilar lymphnodes enlarged by tumor involvement

the presence of severe dyspnea, the percussion note and breath sounds may be unaltered Dyspnea is presumably due to impaired alveolar ventilation and oxygen exchange By x-ray diffuse spread of malignant tumor appears as linear shadows beginning near the hili and progressing peripherally Fine miliary shadows are usually seen scattered and interspersed along the linear shadows; they may coalesce into larger densities Eventually one may find diffuse generalized mottling throughout the lung fields To distinguish diffuse malignant disease from pulmonary congestion, miliary tuberculosis, sarcoidosis, berylliosis, and other disturbances which create widespread granulomatous changes is often impossible radiologically

Pulmonary adenomatosis shows one or more large areas of soft homogeneous infiltration not unlike inflammatory solidification, widely scattered, patchy, nodular areas of infiltration or a combination of the two This disease, too, is likely to be confused with an inflammatory process

TUMOR OF MEDIASTINUM

The most common benign tumors in the mediastinum are dermoid cyst, neurofibroma, substernal goiter, and thymoma Primary malignant disease is most likely lymphoma, secondary disease may represent direct extension from carcinoma of the lung or metastasis from tumor elsewhere. Except for the constitutional symptoms attributable to any malignant process, the manifestations

are chiefly those created by pressure on or invasion of surrounding structures. Obviously, changes peculiar to a particular tumor may also be evident such as, for example, those of thyrotoxicosis in substernal goiter, or myasthenia gravis, which is sometimes associated with thymoma.

Symptoms

The patient usually complains of dyspnea and cough Paroxysmal or constant, dull, or boring pain is likely as a result of pressure or erosion; where it is felt is a matter of the structure involved.

Signs

These are dependent on the size and location of the lesion One may find hoarseness, displacement of the trachea, dulness beneath upper sternum, evidence of total or partial bronchial obstruction, or indications of impaired venous return, such as distention of veins, cyanosis, perhaps edema of neck, face, upper thorax, and arms and, sometimes, collateral dilatation of thoracic and abdominal veins

X-Ray Findings

Benign tumor or cyst in the mediastinum appears as a rounded or ovoid, sharply defined mass Dermoid and teratoma are most likely in the anterior mediastinum and may show calcification, neurofibroma is posterior Enlarged or aberrant thyroid is high in the thorax close to esophagus or trachea and may cause displacement or indentation of either Thymoma is also high but more likely in close relation to the aorta Lymphoma is seen as nodular or lobulated enlargement of mediastinal or hilar nodes.



FIG. 27 11 Substernal goiter causing widening of superior mediastinal shadow to right Extension of shadow toward left is partly attributable to displacement of mediastinal structures



Fig 27 12. Lymphoma Lobulated enlargement of hilar and mediastinal nodes Scattered areas of density due to pulmonary involvement in both lung fields.

Aortic aneurysm is the disease most likely to be confused with mediastinal tumor. Their differentiation has been discussed in Chapter 17.

EMPHYSEMA

Pulmonary emphysema is inflation and dilatation of the alveoli. From the *pathologic* standpoint the term is restricted to cases in which there are distention of vesicles, loss of elasticity, and actual destruction of the septa between adjacent alveoli, resulting in the formation of larger sacs by union of smaller ones. This is obviously an irreversible process, for once the walls have been destroyed they cannot be reconstructed This type occurs in two forms:

1. Hypertrophic or large-lunged emphysema.
2. Senile, atrophic, or small-lunged emphysema. Here the alveolar walls are destroyed but distention is lacking.

From the *clinical* standpoint, the term "emphysema" is also used to denote conditions in which part or all of the pulmonary parenchyma becomes distended but without appreciable alveolar structural changes (*See below:* Compensatory and obstructive emphysema.)

Chronic Hypertrophic Emphysema

When well-advanced, this is associated with *barrel chest.* Many cases appear without known cause. One theory explains them as due primarily to loss of pulmonary elasticity with resultant inability to expel air. Since inspiration is more forceful than expiration, more air is drawn in than can be expired and the lungs become more and more distended To accommodate for the greater pulmonary volume, barrel chest develops. Another theory is that emphysema is due to structural skeletal changes brought about by osteoporosis Fixed ex-

aggeration of the posterior thoracic curve of the spine leads to *increase in the* anteroposterior diameter and hence the capacity of the thorax, resultant greater negative pressure causes the lungs to overexpand and become emphysematous. *Both explanations* may be correct Hypertrophic emphysema also occurs in long-standing asthma and chronic bronchitis, presumably as a result of constant overdistention of *alveoli.*

In the various forms of chronic pulmonary disease, such as fibroid phthisis and fibrosing pneumonitis, localized areas of emphysema may appear in the non-diseased portions of lung.

Symptoms

Progressive dyspnea, eventually disabling, is the *predominant symptom. In* contrast to that due to most other causes, it may be less distressing with the patient supine Cough and expectoration may result from associated chronic bronchitis During the winter months these symptoms are aggravated and the patient is especially susceptible to acute respiratory infection.

Signs

Changes of Thoracic Cage. The posterior curve of the spine is exaggerated, the neck appears shortened, the shoulders are high and stooped, and the intercostal spaces and costal angle are widened Respiratory movement of ribs and diaphragm is limited, the latter is low.

Increased Resonance.

Extension of Lung Margins. On percussion, the area of cardiac dulness is diminished or obliterated. The upper border of liver flatness is one or two interspaces below its normal position Pulmonary resonance extends 2 cm or more below the usual position in the back and axillae and 1 or 2 cm higher than normal at the apices Auscultation helps to substantiate these findings

Diminished Tactile Fremitus.

Changes in Sounds. The breath sounds are always diminished The inspiratory phase is likely to be feeble or absent, the expiratory phase feeble, prolonged, and low-pitched Whispered and spoken voice are diminished, the former may not be heard at all. In pure emphysema, rales are not present

Diminished Heart Sounds. Because of the overlying and distended lung, heart sounds are often very difficult to hear

X-Ray Findings

The lung fields are large and bright, the markings faint, and the diaphragm low. On lateral projection, enlargement of the thorax is apparent and the sternum may bulge anteriorly. By fluoroscopy one sees limited motion of the diaphragm and slow contraction of lungs on expiration If the patient is instructed to exhale forcibly with his mouth open, failure of the diaphragm to move with the normal degree of promptness is observed

Other features of advanced emphysema are: polycythemia, lowered vital ca-



Fig 27 13 Chronic hypertrophic emphysema Increased radiolucency, diminution of lung markings, and low position of diaphragm As is often but not necessarily the case, heart shadow is small Horizontal line below left leaf of diaphragm represents fluid level in stomach

pacity, clubbed fingers (occasionally), and pulmonary vascular hypertension indicated by increased pulmonary second sound and, on x ray, dilatation of pulmonary vessels. Severe emphysema is one of the causes of cor pulmonale and may terminate in right-sided heart failure. However, many cases die of manition presumably based on impaired oxygenization without any clinical or pathologic signs of heart failure

Diagnostic Pitfalls

Obstructive Emphysema. (*See below.*)

Pneumothorax. Here one finds hyperresonance, feeble sounds, and diminution or absence of tactile fremitus, but pneumothorax is usually unilateral, causes displacement of neighboring organs, and is frequently associated with hydrothorax

Pulmonary Tuberculosis. Since tuberculosis can coexist with chronic bronchitis and emphysema it must always be looked for Its signs may be masked. X-ray and the usual special studies for tuberculosis are essential

Mediastinal Tumor. Tumor or aneurysm, by partially obstructing the trachea or a primary bronchus, may produce emphysema and be masked by its signs. Evidence of pressure on other structures and x-ray findings establish diagnosis.

Senile Emphysema

Occurring chiefly in old people, senile emphysema is one of the changes attributed to advancing age. The lungs show loss of elasticity, destruction of air sacs, and probably an increase of residual air, but distention as seen in hypertrophic emphysema is not striking.

Dyspnea may be complained of but is not pronounced Scantily or profusely productive cough secondary to bronchial infection may be evident Lungs are somewhat hyperresonant but not appreciably distended, and tactile fremitus is diminished Breath sounds are diminished but lack the changes in quality encountered in hypertrophic emphysema Rales are dependent on the degree of bronchitis. Thoracic kyphosis, narrowed intercostal spaces, and decreased respiratory motion may be observed As in hypertrophic emphysema, x-ray will show bright lung fields and limited diaphragmatic movement but the changes are less pronounced.

Compensatory Emphysema

When ventilation of a sizable amount of lung parenchyma is impaired, uninvolved portions compensate by becoming overactive Although "compensatory" and "complimentary emphysema" are the terms generally used to indicate this process, they are actually misnomers, since neither appreciable impairment of elasticity nor alveolar structural changes occur Many authorities prefer to call it *compensatory distention*

Depending on the cause, an entire lung, one or more lobes of both lungs, or small scattered areas, may show this phenomenon It is most pronounced in large pleural effusion or pneumothorax, massive atelectasis, widespread lobar pneumonia, and pleuropulmonary contraction due to fibrosis The only symptoms present are those of the underlying disease

Signs

If the compensatory activity occurs in small scattered patches as in generalized tuberculosis or chronic fibrosing pneumonitis, it will not be apparent on physical examination X-ray will show increased radiolucency of the distended lung segments, ventilatory movements are normal When one or more relatively large areas are compensating for loss of function elsewhere one will find:

Increased Respiratory Motion. This is evident only when activity of one lung is grossly impaired, as in large pleural effusion or pneumothorax; contrast between the active and inactive sides will be obvious.

Hyperresonance.

Slightly Increased Tactile Fremitus.

Exaggerated Breath Sounds.

As in hypertrophic emphysema, the distended lung may depress the diaphragm, extend over the heart, and otherwise alter the normal relationship of intrathoracic structures.

Obstructive Emphysema

This develops when impaired outflow of air during expiration causes distention of the alveoli but no structural changes of their walls. Again, use of the term "emphysema" is misleading; *obstructive distention more accurately describes* the alveolar status.

Generalized pulmonary involvement is most apt to be found in acute bronchitis, or acute or chronic asthma. *Here, expiratory discharge of air from the* alveoli is hampered by spasm or inflammation of bronchioles and retained secretions. The signs are comparable to those found in hypertrophic emphysema but *the breath sounds are partially masked by wheezes and rales. In acute bronchitis* or an acute attack of asthma, the process is reversible; the lungs regain normal status once the episode is over In chronic asthma or frequent acute attacks, *alveolar damage may develop and eventually create the picture of hypertrophic* emphysema

Localized involvement is most often due to "trapping" of air distal to the lesion in partial obstruction of a bronchus as by tumor or foreign body If a major bronchus is involved, one will find signs comparable to those of hypertrophic emphysema over the affected pulmonary segment, probably with a local respiratory wheeze. When a smaller bronchus is the site, the emphysematous segment is not large enough to produce signs, but local wheeze is likely In either case, x-ray will show hyperaeration; sometimes *it will be detected only* during fluoroscopy or by comparison of films taken at full inspiration and expiration



Fig 27 14 Obstructive emphysema due to non radiopaque foreign body in primary bronchus.

A Film taken at height of forced inspiration Emphysema of left lung indicated by increased volume and radiolucency, diminished markings, and homolateral flattening of diaphragm

B Film taken at nadir of forced expiration Trapping of air on left indicated by lack of change in lung field Right lung shows normal deflation Heart and mediastinal shadows have shifted to right as a result of diminution of *right lung volume* A bean totally obstructing lower and partially obstructing upper lobe bronchus was removed at bronchoscopy

If the obstruction is corrected, the affected segment of lung returns to normal; if it becomes complete, the picture changes to that of local atelectasis with, later, superimposed infection

PULMONOCARDIAC FAILURE

This term is applied to a syndrome often encountered in association with severe deformity of the thoracic cage, especially kyphoscoliosis Absolute and relative reduction in lung volume with resultant diminution of vital capacity and oxygen exchange is thought to constitute the fundamental difficulty. Increased pulmonary arterial pressure is also a factor It is characterized by progressive dyspnea and usually ends in premature death from sudden cardiac failure. The accepted therapeutic measures are not as effective as in other forms of heart disease Persons so afflicted are especially susceptible to respiratory infection and to respiratory depressants and anesthetics

Symptoms

Breathlessness develops early, increasing with time and progression of the skeletal deformity. At first it does not hamper ordinary activity, but in early or middle adulthood gradually reaches the point of being present without effort. The patient may be more comfortable lying flat or in hyperextension, than sitting upright. Eventually he becomes subject, following little exertion, to superimposed episodes of acute paroxysmal dyspnea associated with palpitation, weakness, and perhaps syncope Any such attack may be fatal within hours or days.

Signs

During the early stages, no signs are evident in the heart or lungs, although the former may be poorly heard because of the abnormal shape of the thorax and consequent distortion of the usual intrathoracic relationships Pulmonic second sound may be relatively increased Cardiac enlargement cannot be detected, although right ventricular hypertrophy may be suggested by electrocardiogram During an acute attack, one finds tachycardia, often as high as 150, but no other changes, except perhaps greater increase of pulmonic second sound As a rule, even in the late stages, evidence of right heart strain or failure is not prominent, as in chronic cor pulmonale due to other causes. *Post-mortem* examination, however, will usually show some degree of right-sided hypertrophy and dilatation.

CHRONIC FIBROSING PNEUMONITIS

Chronic fibrosing pneumonitis, sometimes called *chronic interstitial pneumonia*, is not a clinical entity but occurs as a complication of some other disturbance, such as prolonged inhalation of certain dusts, bronchiectasis, tuberculosis, pulmonary abscess, or untreated empyema. It is characterized by progressive fibroid changes which begin in the peribronchial or interstitial tissues or the pleura, and eventually involve all of these structures Depending on the

cause, the change may be limited to one or more segments or appear diffusely throughout the lungs. In tuberculosis, and less often following bronchopneumonia, one may find smaller patchy areas.

Symptoms

These vary with the stage of the process which develops slowly over a long period. Onset is insidious Progressive cough and dyspnea are the key symptoms. The latter may eventually become totally incapacitating Unless there is associated bronchiectasis or abscess, cough is not profusely productive. Hemoptysis is rare Constitutional response depends on the nature of the underlying cause.

Signs

If the lesions are small and scattered, physical examination may show no appreciable local changes A large well-defined area of damage is likely to manifest signs dependent on the increased fibrosis and impaired ventilation, such as dulness, bronchovesicular, rarely bronchial, breath, voice, and whisper sounds, and fine or medium moist rales. Extensive pleural thickening will increase the dulness, diminish the tactile fremitus and sounds and, if very pronounced, distort the thoracic cage Contraction of lung and pleura, when unilateral, may pull the heart and mediastinal contents toward the lesion. With extensive parenchymal damage, unaffected areas show the signs of compensatory emphysema

X-Ray Findings

The picture is one of mottled irregular patterns of density with interspersed patches of emphysema and atelectasis. Its cause cannot be identified by x-ray since it may be due to any chronic inflammatory process.

PNEUMOCONIOSIS

Various substances in dust or near-dust form, if of small-particle size, highly concentrated in the environment and inhaled for a sufficiently long period, will create pathologic changes in the lungs which vary according to the offending agent. Some are serious, others are not *In certain instances (for example, coal miner's disease and silicosis) an added danger is the likelihood of superimposed pulmonary tuberculosis* The hazards involved are sources of increasing concern and study in the field of industrial medicine.

Symptoms

Depending on the causative agent and extent of the process, the patient may have no complaints or develop progressive dyspnea, often becoming totally incapacitating, cough, and thoracic pain due to local pleuritis. Systemic response may reflect poor oxygenization or superimposed tuberculous or non-specific infection Cough, dry at first, usually becomes productive of sputum which generally shows no special characteristics. In coal miners and perhaps graphite workers, it is likely to be slate-colored or black due to the presence of pigment-

bearing cells. In asbestos workers it may show, microscopically, so-called asbestos bodies detectable by *special staining* methods.

Signs

Often local examination reveals very little, even though extensive damage is indicated by x-ray, *or one may* find various combinations of signs reflecting fibrosis, infection, bronchiectasis, atelectasis, and compensatory emphysema. In the late stages the indications of right heart failure are likely.

Carbon

Deposition of carbon (*anthracosis*) is seen in most city dwellers, in coal miners, and, excessively, in certain other industrial workers, especially those using graphite. Ordinarily, *this is a benign* agent, causes no trouble, and is discovered only *post mortem*. Most of the pigment is taken up by *phagocytes*, passes through the lymph channels, and is deposited in the pleura and peribronchial and mediastinal lymphnodes When exposure is overwhelming, peribronchial and interstitial fibrosis develops and the lymph channels *may* become *plugged with* pigment Resultant impaired ventilation causes gradually increasing dyspnea and eventually death from right heart failure

In coal miners, trouble formerly attributed to anthracosis is now regarded as a response, not to carbon alone, but more to the complex mine dust which includes the silicates, silicon dioxide (quartz) and carbon

Iron and Tin

These metals and perhaps others create changes which, except when advanced, rarely impair health or produce symptoms or signs Following prolonged inhalation of fine iron particles, x-ray will show striking diffuse mottling resembling that of miliary tuberculosis, diffuse carcinomatosis, and sarcoid Tin inhalation causes a characteristic x-ray pattern—minute, very dense, discrete shadows throughout the lungs

Silicon

Silicosis is a specific response to silicon dioxide encountered whenever finely divided sand is used, as by foundry workers and granite cutters The predominating picture is the so-called *onion skin* nodule comprised of whorls of collagen and fibrous tissue replacing normal lung tissue Physical examination is usually negative. X-ray changes are striking—slight accentuation of reticular network and enlargement of hilar nodes followed by development of small, discrete nodules throughout the lungs and, eventually, large circumscribed masses reflecting coalescence of the smaller nodules *Superimposed tuberculosis is common*. In its absence the patient may live for years without trouble but ultimately develops the picture of chronic pulmonary impairment.

Other Harmful Substances

In prolonged inhalation of bauxite, certain silicates such as asbestos, diatomaceous earth and some talcs, and perhaps of cotton and sugar cane fibers, the



Fig 27 15 Silicosis. Diffuse mottled infiltration of both lungs in a man who worked as a granite cutter for 30 years.

pathologic picture is somewhat variable but in general one finds extensive peribronchial and interstitial fibrosis with varying degrees of local or general emphysema, secondary atelectasis—especially characteristic of asbestos—and spontaneous pneumothorax—especially characteristic of bauxite and diatomaceous earth Symptoms, physical and x ray signs, and degree of health impairment depend on the character and extent of the pathologic changes.

PULMONARY RESPONSE TO CHEMICALS

Such agents as phosgene, fluorine, chlorine, strong acid and nitrous fumes, *if inhaled in sufficient quantity, will create severe acute pulmonary edema,* sometimes immediately, sometimes as late as 12 hours following exposure. Death within a short time is likely but it is now believed that some patients who have received what might have been a lethal dose have been saved by prompt initiation of positive pressure oxygen therapy Complete recovery usually occurs in non-fatal cases but it is possible that the patient will end up with chronic progressive bronchopulmonary disease The latter may also be the end-result of repeated exposure to small quantities of any of these agents.

Beryllium

Although pulmonary changes are usually predominant, many authorities regard beryllium poisoning as a general constitutional disease In the *acute* form which follows inhalation of high concentration of certain beryllium compounds, the picture is not unlike that of atypical pneumonia. In fact, the disease is usually misdiagnosed as such unless a history of recent beryllium exposure is obtained Cough, dyspnea, substernal pain and sometimes fever appear quite suddenly. One may find no appreciable pulmonary changes, signs of



FIG 27 16 Chronic berylliosis Diffuse, tiny, miliary densities, which in some areas have become confluent, evident throughout both lungs Patient had worked on fluorescent tubes for several years

bronchitis, or an equivocal picture—slight dulness, altered breath sounds or rales X-ray shows patchy density simulating atypical pneumonia Although clinical recovery within a month or so is the rule, x-ray changes are likely to persist for a year or more

The *chronic* form may be due to longer but probably less intense exposure, or be a late response to acute exposure The latent period may be as long as 10 years The delayed reaction is thought to be due to constant feeding of beryllium into other organs from storage centers which laboratory studies suggest are in the bones. Pulmonary symptoms vary from none to slowly progressing dyspnea, hacking non-productive cough, and often substernal pain. Total respiratory incapacity is a likely end-result Anorexia, weakness, fatigue, and cachexia may result, presumably from liver involvement or inability to utilize proteins Examination of the lungs is often surprisingly barren in spite of extensive disease evident by x-ray. In the later stages, however, one may find the changes encountered in other forms of chronic pneumonitis. X-ray shows diffuse miliary infiltration, often with hilar lymphnode enlargement, and perhaps peripheral emphysema and areas of pneumothorax The picture is readily confused with that of miliary tuberculosis, diffuse carcinomatosis, or sarcoidosis. Death from progressive pulmonary disease and cachexia has occurred in about 25 per cent of established cases. Currently, many others are totally or partially incapacitated by pulmonary disease and, as far as is now known, will probably continue to deteriorate

LIPOID PNEUMONITIS

Repeated seepage into the lung of oil—most likely liquid petrolatum—may produce a process easy to confuse with chronic pulmonary infection or tumor. Occasionally encountered in a healthy adult after prolonged use of oily medicaments in the nose, the syndrome is more common in infants, debilitated persons, or those with impaired palatal, swallow or cough reflex, due to persistent use of oil in the nose or as a cathartic. At first oil which has seeped down into the bronchioles and alveoli is removed through the lymph channels by mononuclear phagocytes. But as more collects, the lymphatics become blocked, the alveolar walls and spaces are filled with oil-laden monocytes, and fibrous proliferation of interstitial tissue eventually develops. Ultimately a mass composed of fibrous tissue, trapped oil and oil-laden cells is formed. In ambulatory persons, the oil gravitates to the lung bases, in infants and bedridden adults, to the posterior segments.

There is no characteristic picture. The disease can be asymptomatic or marked by non- or scantily productive cough. Pain is rare. Physical signs vary from none to evidence of local infiltration or definite solidification. The sputum may contain intra- or extracellular oil droplets. If these can be identified chemically or



FIG. 27.17. Lipoid pneumonitis in a man age 50. No symptoms or signs. Lesion discovered on routine fluoroscopy by Dr. Howard B. Sprague. Nodular mass evident in left lung at level of hilus. Lateral view showed it to be in superior segment of lower lobe. Lobectomy was performed. Preoperative diagnosis: Probable carcinoma. Anatomic diagnosis: Lipoid pneumonitis. Subsequent questioning of patient brought out that without the knowledge of his physicians he had been in the habit, for 20 years, of instilling oil drops into his nose at bedtime. Posterior rather than basal site of lesion is perhaps explainable by the fact that he lay on his back during and for some time after each instillation.

by staining methods as mineral oil, the diagnosis is almost certain. Recurrent attacks of bronchopneumonia with cough, thoracic pain, and high fever are likely

X-ray changes are out of proportion to clinical signs. At first increased markings in the lower or posterior lung fields are observed, later, linear and nodular infiltrations, and, finally, definite consolidation A rather typical granular pattern of the infiltration is especially suggestive In the adult, the lesion is uni- or bilateral, basal, and when nodular, easy to confuse with tumor Infants characteristically show posterior symmetrical infiltrations extending outward but not reaching the periphery.

The disease should be suspected in any person with indications of obscure chronic pulmonary disease, especially when x-ray signs are out of proportion to clinical signs, repeated attacks of acute bronchopulmonary infection have occurred, and a history of prolonged inhalation or ingestion of oil can be obtained

SARCOIDOSIS

This disease, readily confused with other chronic pulmonary disturbances, especially tuberculosis, is of unknown etiology. It pursues the course of a low-grade generalized infection affecting many parts of the body, most commonly the skin, lymphnodes, spleen, eyes, lungs and bones It may last for years, involvement of one structure or tissue beginning while healing is taking place in another All the parts noted are not necessarily diseased in the same patient Constitutional symptoms are not striking and may, in fact, be absent.

The cutaneous lesions, occurring chiefly on the face, nose, ears, eyelids, and extremities, appear as small, disseminated, red or violaceous nodules or larger nodosities Lymphnode enlargement, generalized or confined to one or more groups of nodes, is common The spleen may be slightly to moderately enlarged The characteristic eye change is chronic uveitis with nodules in the iris and cellular deposits on the posterior surface of the cornea, adhesions of iris to lens may be found.

Pulmonary disease is rarely attended by important symptoms or physical signs In fact, these are usually so minimal that trouble is not suspected unless, for some reason, an x-ray is taken If the superior mediastinal or peribronchial lymphnodes are markedly enlarged, cough, submanubrial dulness, and perhaps dulness and bronchovesicular breathing between the scapulae may be observed Sometimes cough, dyspnea, wheezing, rales, and other indications of chronic diffuse bronchopulmonary impairment are present Rarely, cardiac enlargement and eventual failure occur as a result of myocardial involvement

In the well-established case, serum gamma globulin, serum calcium, and urinary calcium excretion are likely to be increased, renal calcinosis or stones may develop as a result of the hypercalcinuria (*see* Chap 32)

X-ray changes are striking Characteristically, one finds symmetrical enlargement of the hilar, and perhaps peritracheal lymphnodes and, often, diffuse, usually symmetrical, miliary to mottled or nodular infiltration of both lungs.

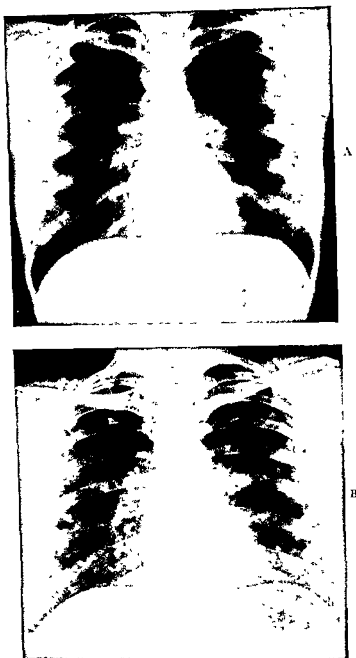


FIG 27.18 Sarcoidosis.

A Slight to moderate enlargement of hilar lymphnodes detected on routine film Woman age 39 with no symptoms

B Four years later Patient had developed persistent cough and dyspnea Hilar lymphnodes somewhat smaller Diffuse mottled infiltration of both lungs Diagnosis confirmed by scalene lymphnode biopsy (Courtesy Dr Kenneth T Bird)

The process may be limited either to the nodes or lung fields or appear to extend from the nodes into the lungs. The picture can easily be confused with miliary tuberculosis, miliary malignant disease, and other causes of diffuse pulmonary infiltration. In contrast to most of these, the x-ray changes may entirely disappear within a few months or years. In a small percentage of cases, cyst-like changes occur in the phalanges of the hands and feet. If found, they may lend support to a diagnosis of sarcoid but they can occur in other disturbances.

FUNGUS INFECTION OF LUNGS

During recent years various fungi have become more generally recognized as responsible for many cases of acute and chronic bronchopulmonary disease. Because of wider and faster travel, fungus infectious are being encountered with increasing frequency in areas where previously they were not found. Among the more common are actinomycosis, torulosis, moniliasis, blastomycosis, coccidioidomycosis, and histoplasmosis.

In some cases, pulmonary disease is the only evidence of trouble, in others, it is part of a generalized progressive infection which affects other organs as well. In general fungi tend to create granulomatous reactions resembling those of productive tuberculosis. In the lungs one finds discrete nodules or confluent areas of infiltration, usually with hilar lymphnode enlargement. Extension to the pleura and chest wall with creation of sinuses may be encountered.

The symptoms, physical signs, and x-ray findings, although varying to some extent with the etiologic agent, closely simulate those of tuberculosis. Fungus infection should always be suspected in any case which cannot be proved due to one of the more common causes such as tuberculosis, malignant disease, or sarcoid. The diagnosis depends on the following features:

1. History of exposure or residence in an endemic area.
2. Character of other manifestations of trouble, such as cutaneous lesions, draining sinuses, or enlargement of liver or spleen.
3. Recovery of the offending organism from sputum, pus, bone marrow, or other structure.
4. Agglutination tests.
5. Skin sensitivity tests.

Coccidioidomycosis is of especial importance because of its tendency to cause one or more thin-walled cavities which may be confused with those of tuberculosis, histoplasmosis, because of diffusely scattered small calcified lesions which are wrongly attributed to previous tuberculosis. In actinomycosis, due to the presence of one or more draining sinuses, the patient is often unnecessarily subjected to multiple operations under the mistaken impression that he is suffering from a chronic pyogenic infection.

OTHER DISEASES AFFECTING THE LUNGS

Diffuse or focal areas of infiltration, fibrosis, or necrosis are sometimes encountered in many other disturbances, including mononucleosis, brucellosis, tularemia, Q fever, psittacosis, erythema nodosum, disseminated lupus erythe-

matosus, periarteritis nodosa, syphilis, and certain parasitic infestations. As a rule, pulmonary involvement is discovered by x ray, physical signs are likely to be absent or minimal. The symptoms are primarily those of the underlying disease Diagnosis can be established only by careful evaluation of the case, bacteriologic, pathologic, and immunologic studies, the course of the disease, and response to various forms of therapy.

28

DISEASES OF THE PLEURA

PLEURITIS

Inflammation of the pleura is usually secondary It may spread from the lung, less often, the pericardium, mediastinum, or a lesion below the diaphragm, or be caused by blood stream infection from some remote focus. It is sometimes seen as a complication of nephritis, rheumatic infection, or gout So called primary or idiopathic pleuritis appearing without evidence of disease elsewhere is to be regarded as a manifestation of tuberculosis unless proved otherwise A picture simulating pleuritis can be caused by direct extension of malignant disease of the lung or metastatic seeding from tumor elsewhere

Acute Fibrinous Pleuritis

Although this may occur as a complication of acute upper respiratory tract infection, especially following undue exposure, it is most often encountered in association with disease of the lung such as pneumonia, abscess, infarct, or tuberculosis

Symptoms

That many cases of acute pleuritis probably occur without producing symptoms or signs is suggested by the frequency with which fibrous pleuritis is found *post mortem* in cases with no history pointing to pleural disease in the past

Onset of the recognizable case is sudden It starts with thoracic pain which varies from a mere "stitch" to a sharp, cutting sensation and is aggravated by inspiration, cough, or change of position It is presumably caused by friction of the two apposed pleural surfaces, or increased tension of inflamed pleura during each expansion of the lung The pain is usually felt in lower axilla or anterior thorax but may appear elsewhere, when diaphragmatic pleura is involved, it is referred to the abdomen, shoulder, or neck Pressure over the diseased area, by restricting thoracic movement, will often reduce discomfort Fever and malaise are the rule but usually, like the pleuritis itself, these are reflections of the underlying infection

Signs

Pleural Friction. This is the outstanding sign (*see* Chap 11) When near the heart, friction sounds must be distinguished from those due to pericarditis (*see*

Chap 23). If pain is severe, respiratory motion may be so limited that friction is not heard.

Restricted Respiratory Movement. With acute discomfort, the patient automatically protects himself by limiting the depth of his respirations, which become rapid, shallow, and often jerky. On the affected side limitation of thoracic movement may be seen and breath sounds are likely to be diminished.

Fixed Position. To ease the pain the patient may persist in lying on one side or the other, sitting upright, or leaning forward. He often presses his hand against the thoracic wall to limit respiratory excursion.

Acute pleuritis is usually easy to recognize but may be confused with myocardial infarction, acute pericarditis, herpes zoster (before the appearance of the eruption), an acute intra-abdominal disturbance, or some disorder of the back causing referred pain. Whenever the diagnosis of acute pleuritis is made, some underlying pulmonary disorder, such as pneumonia, infarction, tuberculosis, tumor, or abscess must always be looked for.

Pleurisy with Effusion

Acute serofibrinous pleuritis may follow a recognizable attack of acute fibrinous pleuritis or, especially when due to tuberculosis, appear unheralded.

Symptoms

When pleurisy with effusion begins during an attack of dry pleuritis, pain decreases because of separation of pleural surfaces by the fluid. Otherwise, the symptoms are the same. Onset is usually insidious with pain, malaise, weakness, cough, and fever; occasionally these develop so rapidly that the picture simulates beginning lobar pneumonia. If ventilation of one lung is impaired by rapid accumulation of a large quantity of fluid, dyspnea will appear, the latter is less likely with slow accumulation, since the other lung has time to compensate for the change.

Signs of Small Effusion

The signs of fluid vary with the amount present, the degree to which it has compressed the lung, and, to some extent, with the position of the patient. Those described in the subsequent paragraphs apply when the patient is upright, the fluid not confined between adhesions or lobes, and the major airway open. Unrestricted fluid tends to accumulate in the most dependent part and will consequently shift when the patient changes position. The x-ray shadow will vary accordingly. Physical signs may change somewhat but rarely enough to be helpful. This statement does not apply to the presence of both free fluid and air, here readily detectable alteration of signs will be found if the patient shi[illegible]

tic[illegible] be detected by x-ray. Fluid somewhat in excess of this quantity will create the following signs at the base:

Dulness to Flatness. This is confined to a narrow zone above the level of the tenth thoracic vertebra

Diminution of Respiratory Excursion.

Absent Tactile Fremitus.

Diminished Breath and Voice Sounds.

Signs of Moderate Effusion

A still greater amount of fluid compresses and displaces upward the lower part of the lung, there are three groups of signs corresponding to. (1) zone of fluid, (2) zone of atelectatic lung, and (3) zone of normal lung

Over Zone of Fluid. The signs here are similar to those indicated above but perhaps more pronounced Flatness may extend as high as the angle of the scapula and be limited to the back If fluid increases, flatness spreads into the axilla and perhaps anteriorly, as far as the sternum, its upper border is then highest in the axilla and curves downward posteriorly and anteriorly, sometimes rising sharply as it reaches the sternum or spine (*S-curve of Ellis*) Palpatory percussion gives a distinct sense of resistance Fremitus is absent, breath sounds are diminished bronchovesicular or absent, voice sounds, diminished or absent

Over Zone of Atelectatic Lung. Here one finds signs resembling those of solidification. dulness, usually increased tactile fremitus, and bronchial breath and whisper sounds which may be normal, increased, or diminished Egophony is the rule.

Over Zone of Normal Lung. The signs here are normal

Displacement of Heart. If the effusion is on the left, the heart is found pushed toward the right With right-sided effusion of moderate size, cardiac shift to the left cannot usually be demonstrated by physical examination, but may be evident by x-ray

Signs of Large Effusion

The whole lung is usually compressed and one will find

Flatness. With the possible exception of a small area at the apex where there may be tympanitic resonance, the whole side of the thorax shows pronounced flatness and sense of resistance

Absent Tactile Fremitus.

Altered Sounds Breath and whisper sounds are diminished bronchial or absent, except at the extreme apex where they may have an amphoric quality Voice sounds are diminished bronchial or egophonic. An important exception to this rule is that if the fluid surrounds solid lung, as for example, in pleural effusion associated with lobar pneumonia, the various sounds are definitely bronchial and likely to be increased.

Displacement of Neighboring Parts. With left-sided effusion the apex of the heart may be pushed upward and as far over as the right mid-clavicular line, with right-sided effusion upward and to the left but to a lesser distance Right-sided effusion may push the liver downward so that its edge becomes palpable.

Trachea and other mediastinal structures are always displaced away from the side of the fluid.

Evidence of Compensatory Emphysema. The lung on the opposite side shows increased resonance and sounds (*see* Chap. 27).

Paravertebral Dulness. With a large or moderate effusion one may find at the base of the opposite lung a small triangular area of impaired resonance extending 4–6 cm. laterally from the spine (*Grocco's triangle*). It does not indicate trouble on that side and has little diagnostic significance.

Signs of Interlobar Effusion

Especially in tuberculosis or pneumonia, fluid may accumulate in the fissure between two lobes It can be seen by x-ray but is rarely detectable on physical examination, although a large amount may cause signs of compression of an adjacent lung segment.

X-Ray Findings

Minimal fluid appears as blunting of the costophrenic angle laterally and posteriorly, by a homogeneous shadow. As the quantity increases, the area of opacity rises, with its upper border forming an S curve continuous with the diaphragm, this is apparent in either the AP or lateral projection A large accumulation causes obliteration of diaphragmatic shadow, compression of



FIG 28 1 Moderate pleural effusion due to metastases from carcinoma of breast Clearly defined homogeneous density in lower third, right hemithorax, laterally it curves upward toward axilla Horizontal line above represents small amount of fluid in the fissure between upper and middle lobes. Left costophrenic angle obliterated by small collection of fluid



FIG 28 2 Massive pleural effusion Homogeneous density in left hemithorax obscuring heart shadow, diaphragm, and essentially all of lung markings Heart and trachea displaced to right

lung with obliteration of its markings, obscurity of cardiac and mediastinal shadows and their displacement toward the opposite side Except when the hemithorax is virtually filled with fluid, or adhesions are present, some change in the shadows will occur with shift of the patient's position since the fluid will tend to seek the most dependent region. Interlobar fluid, depending upon the fissure involved, appears as a circumscribed round or long ovoid area of density That such a shadow is due to fluid rather than tumor or other solid lesion is best determined by a lateral projection in which the former will appear as an elliptical area of density with tapering ends.

Course

When a pleural effusion is absorbed spontantously or is removed, the various pulmonary signs gradually return to normal and displaced structures tend to resume their former positions Friction sounds sometimes reappear temporarily Because of thickened pleura and pulmonary compression the change may take place slowly; dulness and indications of impaired ventilation sometimes remain for weeks, giving the false impression that fluid is still present or has reaccumulated.

The fluid of serofibrinous pleuritis is yellowish in color, has a specific gravity of 1 016–1.020 or higher and, if allowed to stand, will develop a fibrinous clot A few red blood cells and leukocytes, predominantly lymphocytes, are the rule *Grossly bloody effusion or one which reaccumulates rapidly strongly suggests malignant disease of the pleura* The diagnosis can often be established by Papanicolaou or cell-block examination of centrifuged sediment but thoracoscopy may be required.

Diagnostic Pitfalls

Lobar Pneumonia. The indications of lobar pneumonia and pleural effusion, especially when the latter starts abruptly, are quite similar. However, in pleural effusion, the breath sounds are diminished, tactile fremitus is absent, dulness is more pronounced and the organs may be displaced. When the signs of pulmonary solidification in pneumonia are altered by bronchial obstruction due to secretion, a mistaken diagnosis of pleural effusion or empyema is possible. With plugged bronchus and a solid lung, breath sounds are entirely absent and organs will not be displaced, or will be displaced *homolaterally*. With effusion, sounds are diminished but not always absent and organs will be displaced *contralaterally* When lobar pneumonia is complicated by serous or purulent effusion, the physical signs may be confusing because intense (rarely diminished) bronchial breath and whispered voice sounds persist. Dulness approaching flatness, disappearance of tactile fremitus, and presence of well-marked egophony are important signs pointing toward fluid.

Chronic Fibrous Pleuritis. It is often almost impossible to distinguish a small amount of pleural fluid from thickened pleura which creates dulness, diminished breath and voice sounds, and diminished tactile fremitus. In thickened pleura dulness is not as marked, the quality of the breath sounds is less likely to be altered, egophony does not occur, and fremitus is diminished rather than absent

Malignant Disease of Lung. Extensive carcinoma or other large tumor, especially of the lower lobe, gives an absolutely flat note, increased sense of resistance, and often absent breath sounds. Thoracentesis is frequently necessary to distinguish pleural effusion from extensive malignant disease or one of the other disturbances mentioned above

Pericarditis with Effusion. Pleural effusion, especially if confined to the left side anteriorly, is often difficult to differentiate from pericarditis with effusion. The following features suggest the latter

1 Evidence of rheumatic infection

2 Greater respiratory embarrassment and systemic reaction than would be expected with pleural effusion

3 Change in cardiac signs, particularly increased area of dulness to the right or in the supracardiac region, diminution of heart sounds, and paradoxic pulse.

X-ray should clarify the problem.

Disease Below the Diaphragm Subphrenic abscess or enlarged liver from any cause, especially malignant disease or abscess, is apt to elevate the diaphragm. Signs of solidification due to compression of the lung base or indications of associated pleural effusion are likely X-ray is imperative for diagnosis, fluoroscopic observation of diaphragmatic movements being especially important

Defect of the Diaphragm. Eventration, rupture, or hernia of the diaphragm may, because of the presence of stomach or intestine inside the thorax, cause physical signs suggestive of fluid. Gurgling sounds produced by peristalsis suggest the diagnosis, but x-ray confirmation is necessary.

Purulent Pleuritis

Empyema is most commonly a complication of lobar pneumonia but also occurs in bronchopneumonia, as one of the manifestations of septicemia, as a complication of certain other intrapulmonary diseases, particularly abscess, and sometimes by extension of infection from the abdomen or pericardium.

Symptoms

There are no characteristic symptoms, the clinical picture being that of severe infection and as much the result of the underlying disease as of the empyema itself In pneumococcus pneumonia, continuation of trouble for more than 10 or 12 days is suggestive of empyema, its most frequent complication. Sometimes the toxic manifestations may be so mild that, judging from the general picture, the patient has apparently recovered; here purulent pleuritis is suggested by failure of the abnormal signs in the thorax to disappear and by persistence of leukocytosis Empyema complicating streptococcus bronchopneumonia occurs early and is always severe When pleural infection develops in the course of septicemia or by extension from below the diaphragm, the appearance of respiratory symptoms such as cough, pleural pain, and dyspnea, may call attention to the possibility of this complication

Signs

Physical signs are, for the most part, similar to those of serofibrinous effusion There is, however, one important exception in lobar pneumonia, especially of the lower lobe, the solidified lung behind an accumulation of pus may cause the breath sounds and whisper to remain bronchial and intense instead of becoming diminished as they would with fluid occurring over a non-solidified lung Here the signs on which the most reliance can be placed are flatness, pronounced sense of resistance on palpatory percussion, egophony, and, provided it has not been absent from the start, absent tactile fremitus The x-ray picture is similar to that of pleural effusion except that because of fibrinous adhesions change of shadow with shift of position is less likely

When both clinical and x-ray findings fail to provide sufficient basis for a diagnosis of pus, thoracentesis must be performed Pus is most apt to be found where the flatness, sense of resistance, and egophony are most pronounced If there is strong clinical evidence of empyema, and one thoracentesis fails to reveal pus, the procedure may have to be repeated several times A needle of large bore and, for good suction, a large, well-fitting syringe must be used; otherwise thick fluid cannot be drawn out

Empyema can be confused with any of the disturbances mentioned under the differential diagnosis of serofibrinous pleuritis

Encapsulated Empyema

A collection of pus walled off by adhesions is sometimes encountered, most likely peripherally in the lower axilla or in midthorax posteriorly Encapsula-



FIG 28 3 Interlobar pleural effusion Lateral view showing clearly defined, wedge shaped, homogeneous area of density in posterior mid lung field Shadow with diagonal superior margin overlying lower thoracic vertebrae represents a small amount of free fluid at base of pleural cavity

tion between lung and diaphragm or in a vertical channel near the spinal column or along the mediastinal border near the sternum is also possible. When just above the diaphragm, empyema is difficult to distinguish from subphrenic abscess. As a rule, the quantity is not large enough to create physical signs and is discovered only by x-ray which shows a dense, circumscribed shadow.

Interlobar Empyema

Pus in an interlobar fissure rarely occurs independently, being usually part of general thoracic empyema. When not associated with the latter it is almost always secondary to lung abscess *Distinguishing physical signs are lacking* X-ray will show a shadow indistinguishable from interlobar fluid, differentiation between the two depends on the clinical picture

Chronic Fibrous Pleuritis

Fibrous bands between the parietal and visceral surfaces of the pleura are often found *post mortem* There may be only a few light strands, parts of the lungs may be firmly bound to thoracic wall; or one or both pleural cavities may be virtually obliterated. Patches of calcification are likely Often these adhesions have caused no symptoms or signs and there is no history of previous acute or

chronic pleuropulmonary disease. In other cases pleural thickening is sufficiently pronounced to be recognizable clinically. Pleurisy with effusion, empyema, or some chronic pulmonary disease, especially bronchiectasis, tuberculosis, or chronic fibrosing pneumonitis, is the most likely cause.

Symptoms

These usually reflect the underlying lung disease. The patient may complain of pain on deep inspiration or a sensation of restricted respiratory excursion.

Signs

Proportionate to the degree of thickening and contraction, these are usually most pronounced at the base and in the axilla Dulness, diminished to absent tactile fremitus, and diminished breath and voice sounds are the rule At the base, impaired respiratory excursion can usually be demonstrated When thickening and contraction are extreme, one will also find limitation of expansion, retraction of chest wall, sometimes with pronounced thoracic deformity, and displacement of heart and mediastinal structures toward the diseased side Signs attributable to the underlying pulmonary trouble may be present

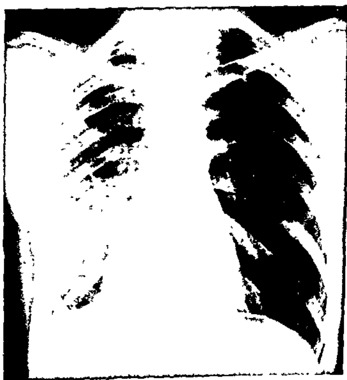


FIG 28 4 Chronic fibrous pleuritis, right hemithorax, due to tuberculosis Hazy density, particularly over lower half, with flattening of hemidiaphragm and obliteration of costophrenic angle Intercostal spaces narrowed Heart drawn to right Some residual parenchymal fibrosis evident in right upper lung field Left lung shows compensatory emphysema (Courtesy Middlesex County Sanatorium, Waltham, Mass.)

X-Ray Findings

Minimal change appears as linear thickening along the thoracic wall with perhaps obliteration of costophrenic angle. Greater change, depending on its severity and extent, will show varying degrees of thickening of pleural shadow, contraction of lung, displacement of heart and mediastinum toward the affected side, and often elevation of diaphragm, contraction of homolateral thoracic wall, and compensatory emphysema of opposite lung, patches of calcification may be seen.

Diagnostic Pitfalls

Pleural Effusion. Since the local signs of pleural fluid and pronounced thickening are similar, one must rely on such *secondary* features as retraction of thoracic wall and displacement of mediastinal structures and x-ray

Malignant Disease. Involvement of the pleura by malignant disease will cause pleural thickening but fluid is likely to be present also.

HYDROTHORAX

Hydrothorax is an accumulation of transudate in one or both pleural cavities Its common causes are congestive failure, constrictive pericarditis, renal disease, liver disease, deficiency states, and other maladies favoring pronounced fluid retention. The symptoms are those of the underlying trouble. Physical signs are identical with those of pleural effusion Hydrothorax is either unilateral or bilateral; pleural effusion is more apt to be unilateral. Unless there are extensive pleural adhesions on the right, a unilateral left hydrothorax can rarely be attributed to congestive failure. Hydrothorax is never accompanied by a pleural friction rub The fluid is of lower specific gravity (1 008–1.015) and cellular content than that of pleuritis.

Uni- or bilateral hydrothorax is also encountered along with a large or small accumulation of intra-abdominal fluid as a complication of fibroma or other benign solid tumor of the ovary (*see Meigs' syndrome*, Chap 33).

PNEUMOTHORAX

Air enters the pleural cavity, causing all or part of the lung to collapse If the pleural surface is free of adhesions the entire lung will deflate (*total pneumothorax*) If, due to some previous disturbance, adhesions are present, only that segment which is not tied down to the thoracic wall will deflate (*partial pneumothorax*); here one will find a walled-off pocket of air between thoracic wall and deflated lung segment.

Total Pneumothorax

Total pneumothorax is most apt to occur from puncture wound or rupture of an emphysematous bleb It is also encountered in tuberculosis, lung abscess, empyema, pneumonia (presumably due to a rupture of a small subpleural abscess) and other bronchopulmonary diseases. However, in the chronic case, partial pneumothorax is more likely because of associated adhesions Rupture of

an emphysematous bleb is often found in a healthy person with no other evidence of pleuropulmonary disease (*spontaneous pneumothorax*) It may be recurrent. Although formerly thought to be due to tuberculosis, it is now no longer so regarded.

Symptoms

Onset is marked by sudden pain in the chest, a feeling of suffocation, severe dyspnea, moderate cyanosis, and sometimes evidence of circulatory collapse. These gradually disappear as the body adjusts to the changes in intrathoracic pressure. This picture is particularly characteristic of spontaneous pneumothorax.

Signs

Restricted Respiratory Excursion. Movement on the affected side is impaired If air in the pleural cavity is under enough pressure, the intercostal spaces may be level with the ribs.

Tympanitic Resonance. Ordinarily the percussion note is tympanitic, but if valvular action at the site of the leak causes air to be continually drawn into the pleural cavity but not expelled, such tension is created that the note becomes muffled and almost dull

Absent Tactile Fremitus.

Altered Sounds. The breath, whisper and voice sounds are diminished and have an amphoric quality Sometimes they are absent

Bell Tympany (*see* Chap 10) The coin test may bring out a clear-cut ringing sound over the pneumothorax in contrast to the dull clank heard over the normal side



Fig. 28 5. Total pneumothorax, spontaneous Arrows point to margin of collapsed left lung Left hemithorax otherwise shows absence of lung markings and increased radiolucency due to free air in pleural cavity. Heart and mediastinal structures displaced to right Prominence of right pulmonary markings reflects compression of the lung

Indications of Compensatory Emphysema on Unaffected Side.

Displacement of Neighboring Structures. Total pneumothorax, particularly if air is present under pressure, will cause greater displacement of the surrounding structures than any other intrathoracic disorder. The heart, mediastinal contents, and trachea are pushed away from the diseased side, the liver downward.

X-Ray Findings

The affected side of the thorax is abnormally radiolucent, lung markings are absent, and collapsed lung is usually observed as a small dense shadow at the hilus. If intrathoracic pressure is elevated the diaphragm is displaced downward, the heart and mediastinal contents away from the affected side.

Partial Pneumothorax

As noted above, this can occur in a variety of *chronic bronchopulmonary diseases*. Obviously, its size depends on the degree to which lung can be deflated by the entering air. The more widespread and extensive the adhesions, the smaller *the segment of involved lung, and hence, the smaller the pneumothorax.*

Symptoms

If a large segment of lung is collapsed, the patient may complain of some pain and dyspnea, but these are not striking. A small pneumothorax is likely to give no symptoms except perhaps transient pain at the time of rupture.

Signs

If the air pocket is small, there may be no signs, diagnosis is made only by x-ray Usually, however, a sufficient segment of lung is deflated and enough air accumulates to produce, at least over a limited area, signs similar to those described above. Compensatory emphysema and displacement of mediastinal structures do not occur unless a large portion of lung is collapsed.

X-Ray Findings

A narrow band of radiolucency around the lung will be observed, or, if adhesions are present, a circumscribed pocket between lung and thoracic wall

Diagnostic Pitfalls

Emphysema. This shows increased resonance and diminished breath sounds, but is a chronic process, usually bilateral, and does not displace neighboring structures Emphysema may be complicated by rupture of an emphysematous bleb, *resulting in pneumothorax*

Pleural Effusion. Air in the pleural cavity, if under pressure, may produce a dull percussion note which, along with diminished sounds and displacement of organs, suggest pleural effusion In pneumothorax, however, *one* does *not* get flatness or the sense of resistance characteristic of fluid, displacement of organs is greater.

Large Pulmonary Cavity. Especially in tuberculosis, the physical signs of a



FIG 28 6 Eventration of left hemidiaphragm. Below elevated leaf (arrows) is a gas-filled segment of colon Heart and mediastinal shadows displaced to right (In this case the abnormal physical signs might well have been erroneously attributed to pneumothorax)

small localized pneumothorax and a large cavity may be confused The breath sounds over a cavity are more amphoric and likely to be louder X-ray will usually clarify the situation.

Defect of Diaphragm. In eventration, rupture, or hernia of the diaphragm, signs resembling those of pneumothorax may be caused by the presence in the thorax of stomach or loops of intestine. Correct diagnosis can usually be made if peristaltic sounds are heard over the thorax but x-ray is often necessary

Myocardial Infarction or Pulmonary Embolism. Because of sudden chest pain, dyspnea and near collapse at onset, pneumothorax, especially when spontaneous, is often mistaken for one of these diseases Physical signs should make diagnosis clear.

Subphrenic Gas. Rupture of a peptic ulcer and certain other intra-abdominal insults may cause gas to collect beneath the diaphragm and give signs suggesting pneumothorax. The differential diagnosis is difficult but can usually be made by careful evaluation of the course of events and x-ray studies

HYDROPNEUMOTHORAX PYOPNEUMOTHORAX. HEMOPNEUMOTHORAX

Pneumothorax can be complicated by the appearance of serous, purulent, or bloody fluid, or, following trauma, blood in the pleural cavity. Conversely, if the pleural space contains one of these fluids, the picture can be complicated

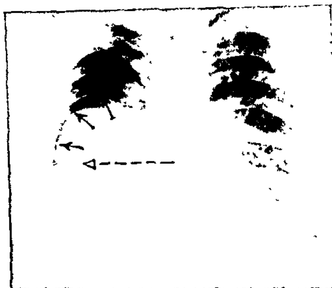


FIG 28 7 Right hydropneumothorax Film taken with patient upright Margin of collapsed lung indicated by solid arrows, fluid level, by broken arrow.

by entrance of air; it can gain access from without, as, for example, during a thoracentesis, or from the lung if the visceral pleura is perforated by trauma or infection.

Signs

In addition to the indications of pneumothorax one will also find:

Succussion Sounds (*see* Chap 11).

Positional Changes of Percussion Note. With the patient upright, a zone of dulness or flatness will be found at the base, tympany above Both zones will shift if his position is chan
pendent part of the thorax,
uncomplicated by air, this
clinically

Metallic Tinkle (*see* Chap 11). This sound may be heard especially at the end of inspiration or after cough but it is not of great importance.

X-Ray Findings

The picture represents a combination of those created by pleural effusion and pneumothorax A clear-cut line of demarcation between the fluid and air will be seen; whatever the patient's position, this line will always be horizontal, since the fluid seeks the most dependent region and the air collects above it.

PNEUMOMEDIASTINUM

Air may reach the mediastinum following operation on the neck, rupture or injury of trachea, bronchus, or esophagus, or therapeutic or traumatic pneumoperitoneum. Sometimes, presumably from a ruptured alveolus, it will leak

Fig 28 8 Chart of physical signs commonly found in various types of pulmonary and pleural disease (*Prepared with assistance of Dr Donald S King*)

CONDITION	PERCUSSION NOTE	TACTILE FREMITUS	BREATH SOUNDS	SPOKEN VOICE SOUNDS	WHISPERED VOICE SOUNDS	RALES	POSITION OF HEART AND MEDIASTINAL STRUCTURES
Normal lung	Resonant	Normal	Vesicular	Normal	Normal	Absent	Normal
Small area of solidification, bronchus open	Slightly dull	Normal or slightly increased	Bronchovesicular or bronchial, usually diminished	Diminished, normal or increased bronchial	Diminished, normal or increased bronchial	Present or absent	Normal
Large area of solidification, bronchus open	Dull	Increased	Bronchial	Increased bronchial Occasional egophony	Increased bronchial	Present or absent	Normal
Solidification, bronchus closed	Dull to flat	Absent	Absent or greatly diminished bronchial	Absent or greatly diminished bronchial	Absent or greatly diminished bronchial	Absent	Normal or slightly displaced toward affected side
Solidification with pleural fluid, bronchus open	Flat	Absent	Intense or diminished bronchial	Egophony	Intense or diminished bronchial	Usually absent Occasionally present	Normal or displaced away from affected side
Solidification with pleural fluid, bronchus closed	Flat	Absent	Absent	Absent	Absent	Absent	Normal or displaced away from affected side
Atelectasis, bronchus closed	Dull to flat	Absent	Absent	Absent	Absent	Absent	Displaced toward affected side

Atelectasis, bronchus open	Dull	Increased	Bronchial	Bronchial *Rarely* egophony	*Increased* bronchial	Present or absent	Normal or displaced away from affected side in compressive atelectasis. Displaced *toward* affected side in postoperative atelectasis
Large lung tumor reaching periphery	Very flat	Absent	*Absent*	Absent	Absent	Absent	Normal or displaced away from affected side
Pleural fluid, small amount, lung *not compressed*	Dull to flat	Diminished or absent	Diminished	Diminished	*Diminished*	Absent	Normal
Pleural fluid, *large* amount, lung compressed	Hyperresonance above, flat below	Absent	Diminished bronchial *or absent*	Diminished bronchial or egophony	Diminished bronchial or absent	Absent	Displaced away *from* affected side
Thickened pleura	Dull	Diminished	Diminished	Diminished	Diminished	Absent	Normal or displaced toward affected side
Pneumothorax, small, lung not compressed	Normal or slightly hyperresonant	Absent	Diminished *or* absent	Diminished or absent	Diminished or absent	Absent	Normal
Pneumothorax, large, lung compressed	Hyperresonant Occasionally dull	Absent	Diminished amphoric Occasionally absent	Diminished amphoric. Occasionally absent	Diminished amphoric Occasionally absent	Absent	Displaced away from affected side

into the interstitial lung tissue and dissect backward along the fascial planes into the mediastinum This phenomenon can follow injury to or operation on thorax or lung, appear especially following cough or strain during some respiratory disease such as influenza, pertussis, or asthma, or occur spontaneously *with* or without strain in an otherwise healthy person, usually a young male. In most cases, air escapes readily into the subcutaneous tissues causing subcutaneous emphysema of the neck, perhaps the face and trunk, sometimes into the retroperitoneal spaces and in perhaps a third of the cases, into a pleural cavity, creating a secondary pneumothorax It is thought that the reverse of the last-named is rarely true, air from a pneumothorax does not ordinarily extend directly into the mediastinum. If it does reach the latter, as, for example, following thoracic trauma or operation, the mechanism is probably that of alveolar rupture with dissection along interstitial pathways

Most authorities believe that trouble is caused only when air fails to escape from the mediastinum and sufficient pressure is thus built up to interfere with circulatory or respiratory dynamics

Symptoms

Symptoms are more likely in cases following thoracic operation or injury, respiratory infection, and spontaneous alveolar rupture. Pain beneath the sternum, often extending over the chest and down the arms, sometimes into the back or neck, and an accompanying sensation of constriction or pressure are the rule They may be accentuated by walking, respiration, movements of the trunk, or assumption by the patient of some particular position, most likely bent forward and to the left In the spontaneous case, onset is abrupt Dyspnea occurs only if there is pronounced mediastinal pressure or pneumothorax

Signs

The outstanding sign is a characteristic systolic to-and-fro sound heard over all or part of the precordium and variously described as *crunching, clicking, crackling*, or *tapping* It may be audible at a distance from the patient, sometimes he himself can hear it In some cases, it varies with respiration or may be heard only with the patient in a particular position, most likely lying on his left side The area of cardiac dulness may be diminished Occasionally precordial tenderness and hyperresonance are observed If air has escaped into the subcutaneous tissues, swelling or crepitation, being obvious, is an important clue to diagnosis

With high pressure in the mediastinum, dyspnea, cyanosis, distention of cervical veins, and perhaps signs of peripheral circulatory failure become evident; this complication is not common. Escape of air into the pleural cavity will create signs of pneumothorax

X-Ray Findings

Air will be seen around the heart or great vessels in the A-P or, more likely, a lateral projection.

The abrupt onset and character and location of the pain may readily lead to a mistaken diagnosis of myocardial infarction, pulmonary embolism, or spontaneous pneumothorax. The association of pneumothorax with pneumomediastinum makes it *imperative* that the latter be *looked for* in any case thought to be primarily the former In contrast to myocardial infarction, electrocardiogram is normal.

The course depends on the cause. Most cases clear up without treatment. Spontaneous cases tend to have recurrences. In severe respiratory infection, death can occur but probably as a result of the disease itself In the rare event that signs of circulatory or respiratory embarrassment develop, withdrawal of air from the pleural cavity (if pneumothorax is present) is indicated; if there is no pneumothorax, direct surgical approach to the mediastinum may be necessary to relieve the air pressure.

The characteristic signs produced in the chest by the more common pleural and pulmonary disorders are outlined in Figure 28.8 It must be understood that this chart is to be used as a guide, not as an infallible standard, for, as we have repeatedly pointed out in the preceding chapters, the clinical picture in any of them is by no means always the same.

29

THE ABDOMEN

EXAMINATION

Despite the emphasis which modern medicine places on x-ray, laboratory tests, and other special methods of investigation, such as cystoscopy and proctoscopy, physical examination still plays an important part in the diagnosis of abdominal maladies The results obtained by the basic procedures are not as exact in the abdomen as in other parts, yet any one of the four may supply essential information Palpation is the most useful, but inspection, auscultation, and percussion are often of great value

Abdominal examination is best performed with the patient lying on his back on a table or firm bed which is narrow and at least 3 feet high On a bed which sags under the patient's weight, the normal curves of his back are altered in such a way that the lower border of the anterior thorax and the symphysis pubis are brought closer together and the vertical dimension of the abdomen is shortened Thus the abdominal structures are crowded together toward the back and are less easy to identify Too low or too wide a bed is awkward for the examiner Complete relaxation of the abdominal muscles is essential, the patient's head should rest on a small pillow and he should be spared the discomfort of a cold room or table, or application of cold hands or instruments

Regional Anatomy

The position of structures in or beneath the abdominal wall is, for convenience, indicated with reference to certain arbitrary divisions The simplest method is to draw a perpendicular and a horizontal line through the umbilicus, dividing the abdominal wall into *right* and *left upper* and *lower quadrants* A more detailed plan divides the surface into nine segments by means of two vertical and two horizontal lines The vertical line on each side extends upward from a point midway between the anterior-superior spine of the ilium and midline, roughly following the outer border of the rectus muscle. The upper horizontal line crosses the abdomen at the lower level of the tenth costal cartilages (the lowest points of the thoracic cage), the lower horizontal line, at or just above the level of the anterior-superior iliac spines. Thus, the upper third is divided into *epigastrium*, right and left *hypochondria*; the middle third, into *umbilical region*, right and left *lumbar regions*; the lower third, into *hypogastrium*, right and left *iliac regions*.

A comb . . . two methods of division is also acceptable The term *epigastrium* . . . described. *Right* and *left upper qua*... umbilicus The lower quadrants ... umbilicus except for that just above the symphysis pubis which ... *suprapubic region.*

When necessary, a point can be more exactly defined by measuring its distance from a skeletal landmark such as the ensiform cartilage or the symphysis

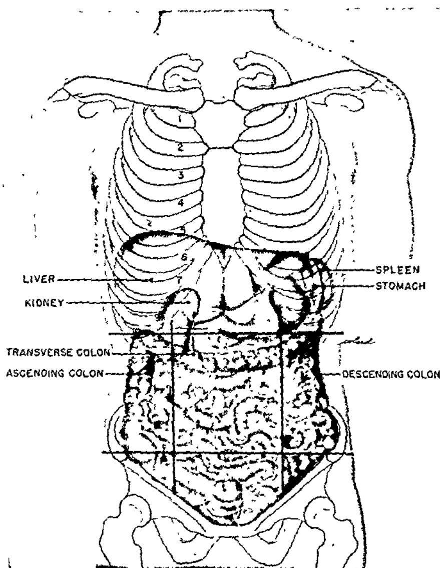


Fig 29 1 Usual positions of abdominal viscera in relation to arbitrary divisions of abdominal wall

pubis, from the umbilicus, from midline, midclavicular line, or one of the axillary lines Tenderness, for example, might be described as in the epigastrium 2–3 cm to the right of midline and just beneath the costal margin, or in the right lower quadrant 3–4 cm. to the right of midline and midway between the level of the umbilicus and the symphysis pubis. *McBurney's point*, important because it is so often the site of maximum tenderness in acute appendicitis, lies 4–5 cm. above and medial to the right anterior-superior iliac spine on a line drawn between this process and the umbilicus.

The *pelvic cavity* is not ordinarily treated as part of the abdominal cavity. This is the region below a plane extending from the upper margin of the symphysis pubis to the promontory of the sacrum

The *inguinal region* or *groin* refers to the groove between the anterior surface of the abdomen and the thigh It extends from the anterior-superior iliac spine to the pubic spine, following the course of Poupart's ligament

Since the abdominal organs are not for the most part as firmly fixed as those in the thorax, their relation to the landmarks cannot be indicated with the same degree of precision In the average person, the position of intra-abdominal structures is approximately that shown in Figure 29.1, but there is considerable variation, depending on the individual's build, muscular tone, and state of nutrition.

Inspection

A tangential light which will accentuate by shadows any unevenness of the abdominal surface is advisable When the patient is lying on his back, the shadow from any but an overhead light is satisfactory; if upright he should stand with his side to the light, not face it When slight movements or variations of contour are being sought, the surface should be studied from different perspectives, often they can be seen only if one's eyes are level with the abdominal surface.

One looks for

1. Abnormal general contour
2. Local prominence or retraction.
3. Cutaneous lesions or other variants.
4. Dilated veins
5. Abnormalities of umbilicus
6. Movements reflecting respiratory activity
7. Movements reflecting peristaltic activity.

Palpation

Methods

Abdominal palpation, the knack of which is acquired only by long practice, is performed chiefly with the cushions and palmar surfaces of the fingers, not with the fingertips. Pressure is applied from the finger joints and wrist, not from the elbow or shoulder The examiner's forearm and hand must be level with or slightly below the abdominal surface, otherwise, in order to avoid use of the

Fig 29 2



Fig 29 3



Fig 29 2 Correct unimanual palpation Examiner's hand and forearm level with patient's abdomen

Fig 29 3 Incorrect unimanual palpation Examiner is palpating from too high a level, with result that his hand and fingers are perpendicular to abdominal surface

fingertips, the hand will be forced into hyperextension at the wrist, its muscles will be under more tension, and flexibility and acuteness of touch reduced If the examining bed is of average height, one should be seated beside it; if unusually low, he should kneel or sit on the floor.

Sudden application of the hand to the abdomen of the average patient will, by inducing muscular resistance in the abdominal wall, prevent satisfactory examination If it is first passed lightly but firmly over the whole surface, the patient will become accustomed to its presence and gradually relax sufficiently to permit deeper and more detailed palpation The hand must be warm and the movements never sudden or rough. Strong pressure with the fingertips or digging into the skin with the nails is inexcusable. Better relaxation is sometimes obtained if the patient's legs are extended; sometimes if legs and thighs are partially flexed, with only his heels resting on the bed. Both positions should be tried

If there is reason to suspect that tenderness or involuntary protective spasm will be found at some particular point, this region should be palpated *last*. The patient's fear of being hurt will be allayed, and he will consequently be less in-

clined to protect himself by involuntarily tensing his abdominal muscles. This approach makes it possible to gage the general sensitiveness of the patient and, by comparison of different regions, to estimate more accurately the importance of whatever tenderness or resistance might be encountered at some particular spot

When slight tenderness is being felt for or the patient is particularly sensitive or apprehensive, a useful procedure is to engage him in conversation on some irrelevant topic, meanwhile watching his face for any expression of pain which might indicate that a tender part is being pressed upon

To feel a structure deep in the abdomen—especially if the latter is large, thick-walled, or tensely held—the fingers of one hand should be placed loosely over the appropriate area and pressure exerted upon them with the fingers of the other The passive hand is more sensitive. The patient is then instructed to take a deep breath, as the abdomen falls in expiration, the hands fall with it Holding the position gained in this way, it may be possible with the next full expiration to advance further and, after a series of such steps, reach the desired objective Obviously, this cannot be done if there is much tenderness, but pure nervous rigidity can often be overcome

Bimanual Palpation. This often provides information not obtainable by the simpler methods The fingers of the left hand are slipped underneath the right or left loin, or a finger is inserted into the rectum or vagina, and pressure is exerted upward toward the right hand, which is meanwhile pressing downward on the anterior abdominal wall above

Ballottement. Useful especially when the abdomen is large or full of fluid,



FIG. 29 4 Ballottement Correct position of examiner's hands and fingers

ballottement may reveal a mass or enlargement of an organ not appreciable on ordinary palpation. The fingertips are suddenly pressed with a quick, stabbing motion into the abdomen and immediately withdrawn; fluid or intervening tissue may be momentarily displaced and a solid structure felt deep in. Sometimes it is possible roughly to estimate the size of tumor or enlarged liver or spleen by partially mapping out its borders in this fashion.

If, when difficulty is encountered in getting the patient to relax, such expedients as having him take a few deep breaths or diverting his attention by casual conversation prove ineffective, palpation can be tried with the patient lying in a tub of warm water. The tub must be long enough for him to lie almost flat. The water should be comfortably warm at the outset, then gradually raised to between 110° and 120°F. Greatest relaxation is usually attained after about 10 minutes' immersion This method has the disadvantage of being awkward for the physician, who must reach down over the rim of the tub and palpate with his hand; it is seldom used.

If relaxation cannot be obtained by any of these procedures, general or spinal anesthesia can be resorted to, but either is rarely justifiable for examination alone However, once the patient has been anesthetized for some surgical procedure, palpation may be most helpful.

Objectives

Distention. If the abdominal cavity is distended with gas or fluid, one obtains an impression of hardness, but to distinguish between the two, other methods are required, especially percussion and examination for fluid wave (*see below*)

Tenderness. This usually denotes inflammation of an underlying structure, stretching of its peritoneal covering, or a tumor. When not detected by ordinary palpation, it can sometimes be elicited by pressing the hand into the abdomen and suddenly withdrawing it (*rebound tenderness*).

Muscle Spasm. Recognized as increased tension or rigidity of the abdominal wall, this may be local or general. It is always of the utmost importance, although often difficult, to distinguish between involuntary or true spasm resulting from inflammation and the voluntary rigidity caused by inability of the patient to relax.

Mass. A mass indicates enlargement of a solid viscus, distention of a hollow viscus by gas, fluid or solid material, or presence of tumor, cyst, abscess, or inflammatory tissue

Peristaltic Movements. Normal peristaltic movements, even though visible, cannot be felt Hyperperistalsis is appreciable as waves of alternate hardening and softening moving beneath the abdominal surface.

Tenderness, spasm, or mass in the abdomen usually points to a disorder of one of the regional structures, as indicated in the outline below. Allowance must be made for the normal variations in relationship between intra-abdominal parts

and the body wall, dependent on type of build, muscular tone, and state of nutrition.

Right upper quadrant	Liver, gallbladder, ascending colon, hepatic flexure, proximal portion of transverse colon, right kidney
Left upper quadrant	Spleen, distal portion of transverse colon, splenic flexure, upper portion of descending colon, left kidney, tail of pancreas
Epigastrium	Stomach, duodenum, left lobe of liver, head and body of pancreas, midportion of transverse colon
Right lower quandrant	Appendix, cecum, proximal portion of ascending colon, right Fallopian tube, right ovary, enlarged or congenitally low right kidney
Left lower quandrant	Lower portion of descending colon, sigmoid, left Fallopian tube, left ovary, enlarged or congenitally low left kidney
Umbilical region	Small intestine, sometimes transverse colon
Suprapubic region	Bladder, female genital organs

Disorder of a regional structure cannot always be assumed, for a sign may be referred from a remote point Tenderness in the right upper quadrant, for example, can be produced by such disorders as acute pleuritis, herpes zoster, or spinal cord tumor Abscess or tumor of an intra-abdominal structure may approach the surface at some distance from its point of origin.

Palpation of abdominal masses is described later in this chapter, of the liver, spleen, and other organs, in subsequent chapters dealing with these structures.

Structures Normally Palpable Beneath Abdominal Wall

When they are normal, the stomach, small intestine, appendix, pancreas, spleen, and bladder cannot be felt The large intestine, for the most part, is not palpable but one can often identify the cecum when it is full of gas, fluid, or solid material, and the sigmoid, especially when it is spastic or full of feces A large collection of solid fecal material in the colon is sometimes mistaken for tumor. Movements of fluid or gas in the stomach can also be felt In most normal persons, with the exception of the obese or muscular, one or more of the following structures are palpable.

Abdominal Aorta. This is felt by deep palpation sometimes above, usually below, the level of the umbilicus, in midline or just to its left Ordinarily only its pulsations are appreciated. However, in a thin person or one with an exaggerated lumbar curve, the vessel lies so close to the abdominal wall that it can almost be grasped between the thumb and fingers; in fact, its course, caliber and motions are so readily appreciable that it is often mistakenly thought to be the seat of an aneurysm. The similarity becomes even more pronounced when one presses on it; here an artificial stenosis gives rise to a systolic murmur and a palpable thrill. The division of the aorta into the right and left common iliacs can sometimes be made out. An easily palpable aorta has expansile pulsation which is present in all normal arteries, not peculiar to aneurysm

Vertebrae. The bodies of the fourth, fifth and sometimes the third lumbar vertebrae are usually palpable at and below the umbilicus, especially in a thin person or one with an exaggerated lumbar curve.

Lower Border of Liver. In most normal subjects the liver cannot be felt, al-

though in a visceroptotic or a person with an unusually wide or narrow costal angle, its lower border may be *felt 1-3 cm. below the right costal margin, or in the epigastrium* Usually deep inspiration is required to bring it into range. Whenever the lower border is palpable, the location of the upper border must be determined by percussion before liver enlargement or downward displacement can be assumed Downward extension of the border is easy to confuse with the not *infrequent congenital anomaly known as Riedel's lobe.* This is a tongue-like downward projection of liver substance which, when present, can usually be felt That it is a local protuberance is suggested by the fact that it is movable, its lateral borders can be made out, and its width is much less than that of an enlarged or displaced liver *The latter can usually be felt all the way* across the right side and sometimes partway *across the left side of the abdomen.* Riedel's lobe can also be mistaken for a distended gallbladder, the right kidney, or a tumor

Right Kidney. In a thin person it is often possible, by bimanual palpation in the right upper quadrant, to feel the lower part or all of the right kidney as it descends during inspiration In a visceroptotic or a *patient with a lax* or *thin* abdominal wall, the organ may be so low, even without inspiration, that it can be grasped between the fingers of the two hands. Rarely in a visceroptotic the tip of the *left* kidney can be felt on inspiration.

Pelvic Organs. The uterus and sometimes the ovaries are palpable, but only on bimanual examination with one hand on the abdominal surface and a finger of the other in the rectum or vagina On the left side a distended sigmoid is often felt bimanually, it is sometimes erroneously regarded as an ovarian cyst.

Muscle Bundle. In a muscular person, a sausage-shaped bundle of muscle tissue is sometimes palpable in the abdominal wall and can be mistaken for an intra-abdominal mass Distinction can be made by having the patient raise his head and shoulders from the table. abdominal muscle, because of contraction, will become harder In a very thin person a comparable mistake may be made by feeling the iliopsoas muscle in the iliac fossa; as a rule this structure is palpable only if inflamed or the seat of an iliopsoas abscess It can be felt to contract and harden if, while lying on the table with his leg flexed at the knee, the patient flexes his thigh.

Study of Abdominal Masses

A mass in the abdomen usually means enlargement of a solid structure such as *congested, fatty, or cirrhotic liver, abnormal distention* of a hollow viscus, tumor, cyst, pregnancy, or an aggregation of inflammatory tissue Sometimes its nature can be determined by physical examination alone—chiefly palpation—but further investigation is often necessary In examining any intra-abdominal mass the following points should be noted·

Position. It has already been indicated that an abnormal finding in any part of the abdomen suggests disorder of a regional structure but that reliance cannot be put on site alone because tumor or abscess starting in one region may have grown in such a way as to be palpable elsewhere.

Size. This may be of considerable significance For example, a mass on the

right side, at once drawing attention to the liver, would, if very large, be more suggestive of malignant disease or leukemia than cirrhosis or congestion.

Contour. A smooth and rounded surface suggests distention of a hollow viscus, a diffusely enlarged solid organ, a cyst or benign tumor; a rough, irregular or nodular surface, malignant disease.

Consistency. A normal hollow viscus cannot be felt. If distended with air, fluid, or semisolid material, it presents a doughy or resilient feel; if infiltrated with malignant tissue, it is hard Cystic tumors have a certain resiliency, whereas malignant tumors are almost invariably hard and irregular.

Tenderness. This must always be interpreted in the light of the patient's threshold of sensitiveness Although local or general tenderness almost always predicates some abdominal disturbance, it is more characteristic of certain troubles than of others For example, an enlarged liver, if tender, is more apt to mean chronic passive congestion or abscess, than cirrhosis or carcinoma Many normal persons show some epigastric sensitiveness; if convincing tenderness is elicited, carcinoma is more likely than ulcer

Pulsation. Visible or palpable abdominal pulsation is common, due to transmission from the aorta Palpable expansile pulsation is usually attributable to normal aorta, rarely, to aneurysm of abdominal aorta or one of its large branches, a highly vascular tumor, or, in the case of the liver, to marked tricuspid insufficiency.

Mobility. The degree of respiratory mobility of an intra-abdominal structure is determined by holding the fingers against its lower border, letting them move with it as it descends with deep inspiration, and measuring, with respect to a fixed point, the number of centimeters they move The stomach, transverse colon, liver, spleen, and kidneys all move downward with inspiration. But normally, except with low liver or right kidney in a visceroptotic subject, this movement cannot be appreciated If stomach or transverse colon is the seat of a tumor, or the liver, spleen, or a kidney is enlarged from any cause, descent with inspiration can often be detected However, failure of a palpable mass to move with respiration does not exclude any of the above-named organs as the seat of the trouble, for it may be fixed by adhesions or, in the case of malignant tumor, by direct extension to a neighboring part The pancreas, retroperitoneal lymphnodes, and structures in the lower part of the abdomen show no appreciable respiratory motion

Relation to Abdominal Organs. Whether or not a mass is an enlarged liver, spleen, or kidney, a tumor of one of them, or in no way connected with any of them, is difficult and often impossible to determine on physical examination. Some light is thrown on the problem, however, by noting whether or not, (1) a groove or space can be made out between what appears to be a mass and the viscus; (2) their respiratory mobility is the same, (3) the percussion note over the mass becomes more or less resonant when the colon is inflated with air (*see below*) X-ray study is of much greater value.

Relation to Abdominal Wall. To ascertain whether a mass involves the skin, one lifts up a fold overlying it; if the skin dimples, it is adherent at that point.

If a tumor is suspected of being in the abdominal wall one can strengthen this impression by grasping a large fold; the mass will appear to be included in the fold, not beneath it.

Peritoneal Crepitus. This relatively rare phenomenon, comparable to pleural friction, can sometimes be appreciated on light palpation as a slight grating or rubbing sensation synchronous with respiratory movement. It is more often audible than palpable. Presumably due to plastic peritoneal exudate, it is most likely to be found over an area of perihepatitis or perisplenitis.

Percussion Note. Percussion is helpful chiefly in distinguishing between a portion of gut distended with gas, and a solid viscus, tumor, or cyst. Distended gut is usually tympanitic, the others, dull or flat, but one must not lose sight of the fact that percussion note of a solid structure can be altered by that of overlying intestine and that a dull note can be created by stomach or bowel distended with fluid or solid material.

Percussion

The indirect method is preferable. The hands are used as in thoracic percussion but the pleximeter finger requires less pressure, and the plexor strokes should be lighter The direct method is used chiefly in testing for fluid wave and estimating degree of resistance

With the exception of the area of liver dulness and flatness and the occasionally appreciable splenic dulness (*see* Chap. 10), the normal abdomen is tympanitic Since tympany reflects the amount of gas in the stomach and intestine, the note is variable and experience is required to distinguish between what is normal and abnormal. Abdominal percussion is used chiefly to determine the presence of·

Gaseous Distention. With excess gas in the intestinal tract or free gas in the abdominal cavity, the note is more tympanitic than normal and drum-like in quality. Sometimes tympany is so pronounced that liver dulness is obliterated (see Chap 10)

Free Fluid. This is determined by finding shifting dulness and a fluid wave (*see below*).

Enlargement of Liver. When the lower border of the liver is palpable a few centimeters below the costal margin, percussion of its upper border is required to decide whether it is displaced or enlarged If flatness begins below the sixth space in midclavicular line, displacement can be assumed; if it is found at this level or higher, enlargement is probable However, these conclusions are reliable only if conditions within the lower part of the right pleural cavity are normal Intrapleural fluid or pulmonary solidification will make the liver flatness appear higher than it actually is; hyperresonance reflecting emphysema or pneumothorax, lower

As a rule, percussion of the lower hepatic border is unsatisfactory because of the tympanitic note created by adjacent intestine.

Diminished Size of Liver. Percussion of its upper and lower borders is sometimes helpful in determining decreased liver size but here too its value is

limited because the zone of dulness or flatness may be poorly demarcated due to a variant in the thorax or gas in the bowel. It is in the patient with serious acute parenchymal disease that percussion of the organ as a whole is most likely to be useful A single observation is of little importance but if day-to-day percussion of the two borders shows them to be gradually receding, shrinkage of the liver—an indication of increasing damage—can be assumed.

Obliteration of Liver Dulness. Usually caused by free gas in the abdominal cavity following perforation of stomach or intestine, this can also be due to extreme distention of the bowel and, during the first few days following surgical operation, to air which has entered the abdominal cavity through the incision. It is not always a reliable sign of perforation

Obliteration of Traube's Semilunar Space. The size of this area (*see* Chap 10) varies with the character and amount of stomach contents: the more gas in the stomach, the larger the area of tympany Obliteration usually indicates either a large pericardial or left pleural effusion, enlarged left lobe of liver, or, less often, moderate to great enlargement of spleen.

Enlargement of Spleen. Some observers believe it possible to demonstrate splenic enlargement by percussion but the consensus is that this cannot be assumed unless the organ is palpable or its x-ray shadow definitely increased

Dilated Stomach. If a severely dilated stomach is filled with fluid one may find dulness, chiefly in the epigastrium to the left of midline, beneath the lower left ribs anteriorly, and extending downward, sometimes even as far as the level of the umbilicus or lower The dulness will not be so evident if gas is also present even though the fluid predominates With the patient supine, the gas, by collecting under the anterior stomach wall, will create a tympanitic note In this event, succussion sounds may be helpful If gas predominates, pronounced tympany will be elicited by percussion over this area

Distention of Bladder. An overfilled bladder can sometimes be percussed as a rounded area of dulness or flatness extending upward from the symphysis pubis It may reach the level of the umbilicus or higher The procedure is not reliable when the abdomen is fat or distended

Enlargement of Uterus or Ovary Dulness in the suprapubic region may be produced by a gravid uterus, large ovarian cyst, or large ovarian or uterine tumor

Relation of Mass to Colon. It is sometimes possible by forcing air into the rectum and distending the colon, to determine by percussion whether an intra-abdominal mass lies anteriorly or posteriorly to the latter. Over a large spleen which lies anteriorly the note will be dull, over a large kidney which would be behind the inflated colon, hyperresonant. Formerly frequently employed, this method has now been superseded by the more accurate ordinary and double-contrast barium x-rays.

Auscultation

In many cases a source of valuable information, abdominal auscultation is all too frequently neglected.

Sounds Due to Peristalsis. In the normal abdomen, one can always hear gurgling, bubbling noises caused by fluid and air moving through the intestines. They show wide variation in frequency, intensity and pitch; what falls within the limits of normal can be learned only through experience.

In *intestinal obstruction* increased peristalsis causes high-pitched, tinkling sounds. They are usually associated with bouts of colicky pain If the obstruction lasts long enough the sounds become diminished, infrequent, and irregular, due to reduced peristalsis resulting from bowel fatigue.

In *intestinal paresis*, the peristaltic sounds are faint, occur infrequently, and may disappear altogether In widespread general peritonitis, because of paralysis, they are usually absent, except in the early stages

Succussion Sounds. Splashing sounds due to the presence of both fluid and gas in stomach or intestine, especially when either is dilated and atonic, can often be elicited by shaking the patient As noted earlier, they may also be heard over the thorax if, through a defect in the diaphragm, stomach or loops of intestine have ascended into the thoracic cavity (*see* Chap 11)

Vascular Sounds. The systolic and diastolic sounds and murmurs described in chapter 12 as occurring over the femoral vessels can sometimes be heard over the iliacs. The murmur of an intra-abdominal arteriovenous fistula may be evident The faint tic-tac sounds of the *fetal heart* are audible in the later months of pregnancy, sometimes as early as the fifth month Where they are best heard depends on the position of the fetus *Placental souffle*, a systolic humming sound occurring just after the mother's cardiac systole, is often evident over the abdomen during the latter half of pregnancy, its exact location depending on the position of the placenta. A *continuous venous hum*, occasionally accompanied by a palpable thrill, can sometimes be detected about the umbilicus, less often above it or in the region of the lower sternum, in obstruction of portal circulation from cirrhosis of the liver or other cause It presumably arises in the underlying veins which have dilated to effect shunt of blood around the portal obstruction

ABNORMALITIES OF ABDOMINAL WALL

Contour

General Retraction. Emaciation is the most common cause Retraction may also occur in the early stages of acute general peritonitis, in meningitis, lead colic, and in diaphragmatic defect, if some of the abdominal organs are displaced into the thorax

Retraction on inspiration, noted especially in the epigastrium, occurs with extreme dyspnea and in cases where there is paralysis of the diaphragm or interference with free flow of air in and out of the lungs, as in tracheal stenosis or severe emphysema

Local Retraction. Contraction of scar tissue in the abdominal wall is the most likely cause

General Prominence. Obesity, lumbar lordosis, and late pregnancy are the

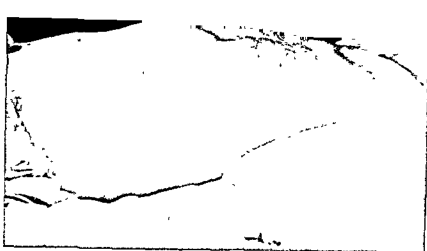


FIG 29 5 Localized swelling in right upper quadrant due to metastatic carcinoma of liver

most common causes Others are ascites, intestinal distention, free gas in the abdominal cavity, greatly enlarged liver or spleen, large tumor or cyst, rickets, cretinism and Cushing's syndrome

Local Prominence. Depending on its location, this may be due to hernia, to abscess or tumor of the wall, or some intra-abdominal variant Specific disturbances to which prominence draws attention are:

Epigastrium	Carcinoma of stomach, dilatation of stomach, marked enlargement of left lobe of liver, pancreatic cyst
Right upper quadrant	Enlargement of liver or right kidney, marked distention of gallbladder
Left upper quadrant	Enlargement of the spleen or left kidney, dilatation of stomach
Right lower quadrant	Appendiceal abscess, marked enlargement of right kidney, cancer of cecum or ascending colon
Left lower quadrant	Marked enlargement of spleen or left kidney, large diverticulum with surrounding inflammatory tissue, cancer of descending colon or sigmoid
Suprapubic region	Distention of bladder, pregnancy, large uterine fibroid, ovarian cyst or large tumor
Inguinal region	Hernia, inguinal lymphnodopathy, psoas abscess, undescended testicle, hydrocele of spermatic cord, cyst of canal of Nuck

HERNIA

Hernia is a protrusion of some of the abdominal contents into or through the abdominal wall at a point where it is weak. The hernial sac is an outpocketing of the parietal peritoneum, projecting into it there is usually a portion of omentum and, depending on the location of the defect, some part of the intestinal tract, rarely, some other intra-abdominal structure. The lesion may be congenital or acquired. In the latter case it is apt to follow strain but there is doubtless a pre-existing local weakness The size of a hernia may vary from a

small nubbin to a huge—10 cm. or more—rounded swelling One which is untreated tends to enlarge. The contents may more or less freely slip in and out of the sac (*reducible hernia*), or become fixed by adhesions or constriction of the neck of the sac (*irreducible* or *incarcerated hernia*). If a segment of intestine is caught in the sac in such a way that its circulation is impaired, the ensuing stasis will result in congestion, edema, and gangrene (*strangulated hernia*).

The patient may be symptom-free or have vague gastrointestinal complaints such as gas, fulness, intermittent pain or colic, and constipation. Hernia appearing suddenly after strain may create acute local pain due to stretch or tear of tissue Strangulation initiates sudden, acute pain and tenderness in the region of the lesion, often with pain referred also to upper or lower abdomen, depending on whether small or large bowel is strangulated. Vomiting and severe colicky pain due to obstruction appear shortly and with them, perhaps, evidence of general circulatory collapse Fever and other signs of peritonitis resulting from necrosis of bowel due to impaired local circulation are late manifestations.

Epigastric Hernia. Usually represented by a swelling in midline at some point

.imes not large

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estinal or stom-

ach wall and appear on the surface as a rounded prominence 1–2 cm in diameter, often visible only when the patient increases intra-abdominal pressure by contracting his muscles. It is usually reducible. Rarely, it presents symptoms not unlike those of peptic ulcer.

Umbilical Hernia. Both the congenital and acquired types are common. The latter is seen particularly in cases of obesity, distention, ascites, and pregnancy As a rule the hernia appears as a soft, rounded protrusion at the umbilicus, rarely more than 1–2 cm in diameter, and easily reducible. A distinct ring-like opening in the abdominal wall can be felt Sometimes, particularly in multiparous old women with poor muscular tone and pendulous abdomens, the omentum, transverse colon, and loops of small intestine are gradually forced out of the opening until a large, soft, rounded mass projects above the abdominal surface. The relatively small caliber of the ring makes incarceration and strangulation common hazards

Inguinal Hernia. This predominates in males because of weakness in the abdominal wall due to passage, in fetal life, of the testicle and spermatic cord through the inguinal canal. In *indirect* or *oblique* inguinal hernia, the sac emerges through the internal ring, extends downward through the canal and often descends into the scrotum An intestinal segment may slide in and out of the sac; swelling disappears when the patient lies down and reappears when he stands or raises abdominal pressure by straining or coughing If, with the hernia reduced, a finger is introduced into the external ring by invaginating the skin and wall of the scrotum along the course of the spermatic cord, a definite impulse can be felt as intestine is forced back into the opening when the patient coughs or strains In *direct* inguinal hernia, which reflects weakness of the posterior wall of the canal, a rounded swelling appears just above and lateral

to the symphysis pubis. It is less likely than indirect hernia to become large or involve the scrotum Occasionally the bladder wall will form the sac's medial wall, creating evidence of bladder irritation, such as frequency and urgency of micturition. Whether an inguinal hernia is direct or indirect sometimes cannot be determined except at operation.

Femoral Hernia. This is more common in women The sac extends downward into the femoral canal beside the vessels, creating a small swelling on the inner aspect of the thigh below Poupart's ligament and just lateral to the symphysis pubis With enlargement, by working its way medially and upward, it may become difficult to distinguish from inguinal hernia. However, it does not produce a cough impulse in the ring This type is particularly apt to strangulate.

Incisional Hernia. Resulting from weakness of the abdominal wall at the point of previous surgical incision, postoperative hernia is most apt to develop in fat or weak-muscled patients and in those who have required postoperative drainage through the incision Common sites are the right lower quadrant, right upper quadrant and, especially in women, the midline above the symphysis pubis The hernia may be small or, in a patient with a flaccid abdominal wall, reach huge size, so that much of the omentum, transverse colon and small intestine lie outside the abdominal cavity

Diastasis of Rectus Muscles

Usually encountered in a person with poor abdominal musculature, the rectus muscles are separated in midline above or below the umbilicus and a definite groove can be felt between them The latter may be just large enough to admit one or two fingers or, in an extreme case, so wide that if the patient stands or strains, loops of intestine protrude through it Through a wide opening, the palpating fingers can reach further than usual into the abdominal cavity and more readily feel the regional structures

Diastasis can be differentiated from a large ventral or umbilical hernia, which it resembles, by the absence of a palpable ring, and the tendency of the protruding structures to return spontaneously to the abdominal cavity when the patient lies down. Strangulation does not occur.

Skin

Eruption. The cutaneous lesions of most of the specific exanthematous diseases and of many dermatological disorders appear on the abdomen The rose spots of typhoid fever—pinkish, slightly elevated macules averaging 2–4 mm. in diameter—have a predilection for this site. Whenever typhoid fever is suspected, the abdomen must be carefully inspected from day to day, because these spots, so characteristic of the disease, are sparse, appear in crops and last only a few days at a time.

Striae. These point to past obesity, ascites, pregnancy, or some other cause of abnormal stretching of abdominal wall.

Scars. Surgical or traumatic scars may be of some diagnostic help, particularly

in a comatose or delirious patient who is unable to give a dependable history.

Abscess. Usually pointing on the surface, this is uncommon except following trauma or operation. In the former it is usually caused by infection and necrosis of a hematoma. Postoperative abscess represents contamination of the surgical wound at the time of operation.

Dilated and Tortuous Veins

These are seen in diseases causing obstruction of portal or caval venous flow. Dilated veins within the wall not appearing on the surface can be detected by infra-red photography.

Determination of the direction of blood flow in such vessels is helpful in differentiating between obstruction in the portal and vena caval systems. Two fingers held closely together are pressed upon a section of vein which is free of branches, then moved apart along its course so that the segment between them *is empty of blood. By lifting one finger at a time while the other remains* pressed down on the vein, the direction from which blood enters the vein can be determined.

In a normal person, venous blood flow in the abdominal wall *below* the umbilical level is *downward* into the saphenous veins; *above* the umbilical level, *upward* into the vessels of the thoracic wall.

Portal Obstruction. Some of the blood normally destined to pass through the liver is shunted through the round ligament and reaches the superficial abdominal veins through anastomoses around the umbilicus. The veins above and below may become dilated but the *direction of flow is normal*. An occasional case of cirrhosis of the liver will show a group of distended veins radiating from the umbilicus (*caput medusa*), its rarity makes it of little importance. Venous hum originating in visible or invisible dilated abdominal veins concerned with the shunt in portal obstruction has already been mentioned (*see above*).

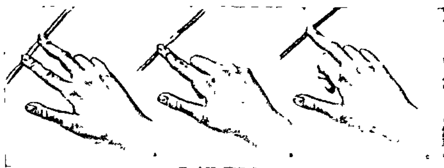


Fig. 29-6. Diagram illustrating method of determining direction of blood flow in a vein.
A. Section of vein between the two fingers has been emptied of blood.
B. Finger on right is removed, vein does not fill.
C. Finger on right is replaced and the one on left removed, vein fills indicating that flow of blood is from left to right.

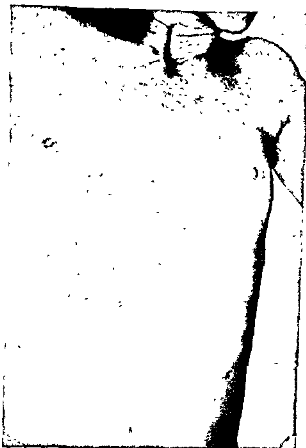


FIG 29 7 Distention and tortuosity of veins of thoracic wall and upper abdomen due to portal and hepatic venous obstruction from hepatoma superimposed on cirrhosis of liver Blood flow above umbilical level is in normal direction (upward) (Courtesy Dr Robert R Linton)

Inferior Vena Caval Obstruction. Some of the venous blood from the lower part of the body is shunted to the superior vena cava by way of the vessels of the abdominal and thoracic walls Here venous flow below the level of the umbilicus is *from below upward*, the reverse of the normal course

Superior Vena Caval Obstruction. Occasionally venous blood from above is shunted to the inferior vena cava by way of the thoracic and abdominal walls obviously the flow is *downward* More likely in superior obstruction, the shunt is into the azygos system through the thoracic veins so that the load on those of the abdomen is not increased.

HEMATOMA OF RECTUS MUSCLE

Spontaneous rupture of an artery in the abdominal wall, usually the inferior epigastric, with resultant hemorrhage between the rectus muscle and its posterior sheath, is occasionally encountered It is most likely in an older woman with arteriosclerotic vessels and weak musculature, and may be initiated by cough, sneeze, or mild external trauma Sudden pain, tenderness and spasm, sometimes accompanied by nausea, vomiting and distention and perhaps by an ill-

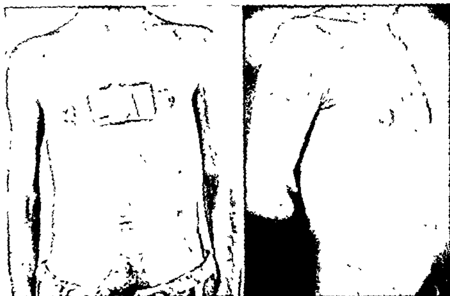


FIG 29 8 Distention and tortuosity of veins of thoracic and abdominal walls due to obstruction of superior vena cava in a patient with bronchogenic carcinoma Blood flow is downward, above umbilical level this is the reverse of normal (Patch covers recent sternal biopsy incision)

defined mass thought to be intra-abdominal, create a picture which is readily mistaken for some acute intra-abdominal insult such as intussusception, volvulus, twisted ovarian cyst, acute appendicitis, or mesenteric thrombosis

The Umbilicus

Projection. Umbilical hernia, or enlargement or distention of the abdomen from such factors as obesity, ascites, and pregnancy, may cause the umbilicus to be level with or protrude above the surrounding surface

Infiltration. Thickening or inflammation may occur in carcinoma or tuberculosis of the peritoneum Hard infiltration secondary to intra-abdominal malignant disease is occasionally encountered

Discharge Intermittent or continuous, serous or seropurulent exudate associated with redness and other evidence of low-grade inflammation is usually due either to fungous or bacterial infection of the umbilicus or, less often, to a patent urachus Rarely, in the latter, the discharge is urinary

Discoloration. Bluish discoloration sometimes occurs in hemorrhage into the abdominal cavity but, being a late manifestation, is not often seen It is most likely to occur following ruptured spleen or ectopic pregnancy Even more rare is bluish discoloration at the time of catamenia due to an endometrial tumor of the umbilicus

Respiratory Movements

Respiratory movements of the abdominal wall become restricted or cease altogether in intra-abdominal disorders causing severe pain such as peritonitis,

in upward displacement of the diaphragm from ascites or other causes of abdominal enlargement, in diaphragmatic paralysis, or restriction of diaphragmatic movement from any cause.

Peristaltic Movements

Peristaltic movements are not usually visible but may be seen in a person with a thin lax abdominal wall, especially if one looks for them with his eyes level with the surface. In chronic pyloric or intestinal obstruction, waves are almost always visible, except in the obese or muscular *The distinction between normal and pathologic peristalsis must be made by palpation or auscultation, inspection is not dependable.* In obstruction the intestinal wall feels firm while the wave is passing; when activity—even though visible—is normal, the wall remains soft and seems to disappear under slight pressure. The movements attributable to chronic pyloric obstruction appear in the upper abdomen and seem to pass from left to right When the small bowel is chronically obstructed near the cecum, waves are seen in the umbilical region and, in a thin person, distended loops are apt to show parallel ridges extending horizontally across the abdomen (*ladder pattern*) If small bowel obstruction is higher, the upper abdomen is distended, waves may be seen at one or two points, but no definite pattern develops In chronic large bowel obstruction definite waves are rarely seen but, proximal to the lesion, segments of distended bowel may, as they contract, harden and rise beneath the surface, softening and receding as they relax Chronic obstruction in the distal colon creates symmetrical abdominal distention, at a higher level, asymmetrical distention

Acute obstruction, in any part of the bowel, creates early distention but peristaltic waves are not ordinarily palpable or visible. At first auscultation will reveal increased peristaltic sounds, later, diminished or absent sounds.

DISORDERS OF THE PERITONEUM

Ascites

An abnormal quantity of fluid in the peritoneal cavity is not a disease entity It may predicate disease of the peritoneum itself but is more likely secondary to a disturbance elsewhere. Its usual causes are:

1. Congestive heart failure, nephritis, nutritional deficiency, or other maladies favoring pronounced fluid retention

2. Interference with return flow of blood from the lower part of the body, as in portal cirrhosis of the liver, Banti's syndrome, Chiari's syndrome, constrictive pericarditis, and high obstruction of the inferior vena cava In portal cirrhosis, the ascites is partly the result of other factors, especially diminution of serum protein.

3. Acute hepatic insufficiency, as in severe acute hepatitis

4. Tuberculous peritonitis

5. Peritoneal carcinomatosis

6. Tumor of ovary Fibroma or other solid ovarian tumor, in some way which is not understood, may cause ascites (and hydrothorax), which will disappear if

the tumor is removed (*Meigs' syndrome*) In carcinoma, ascites can result from peritoneal implants or projection of secreting tumor cells through the ovarian wall Peritoneal implants secondary to benign pseudomucinous cystadenoma create a mucin-like intra-abdominal fluid.

7. Abdominal lymphoblastoma and, rarely, myeloid or other form of leukemia in its terminal stage.

8 Acute general peritonitis Serofibrinous or purulent fluid is present, but rarely in sufficient quantity to be grossly evident

Signs

These depend on the volume of fluid and the pressure under which it exists. The degree of pressure, in turn, depends on the speed of accumulation and the capacity of the cavity Free fluid will ordinarily be discovered earlier in a small thin person than in one who is large-framed or fat; less than a liter is probably never recognized clinically

Abdominal Enlargement. Increased size of the abdomen and tenseness of its wall are almost always the first signs When fluid collects rapidly it is under greater pressure, enlargement of the abdomen is symmetrical, the wall is tense, and leveling or projection of the umbilicus occurs early With slow accumulation, since the tissues have time to stretch, the wall is apt to be less tense and abdominal enlargement not as symmetrical: when the patient is upright, the lower abdomen bulges, when he is supine, his flanks bulge With a large amount

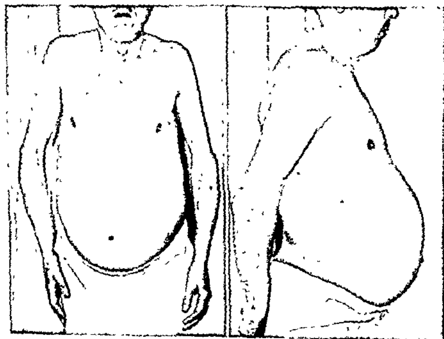


Fig 29 9 Swelling of abdomen due to ascites in a case of hepatic cirrhosis Note projection of umbilicus in lateral view

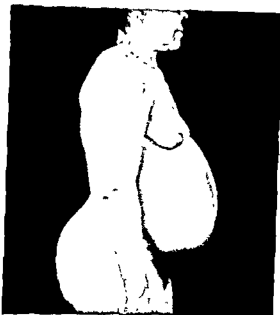


FIG 29 10 Swelling of abdomen due to huge ovarian cyst Umbilicus not protruded (Courtesy Dr Thomas H Green, Jr)

of fluid, irrespective of its speed of accumulation, the abdomen is enormously enlarged, tense, and of globular shape The umbilicus is projected and frequently shows herniation Because of the mechanical handicap of the added abdominal weight, the patient, like a woman in late pregnancy, in order to maintain balance, stands and walks on a wide base.

Shifting Dulness. Since free fluid gravitates to the most dependent regions, the note here is dull The gas-containing loops of intestine float upward and give a tympanitic note. When the patient is supine, the fluid and dulness are in the flanks and suprapubic region, the intestine and thus the tympanitic note are in the epigastric and umbilical areas One cannot be certain of the presence of fluid, however, unless he can demonstrate a shift of the sites of dulness and tympany with change in the patient's position (*shifting dulness*) Lines are drawn on the abdominal surface mapping out the zones of dulness and tympany, first with the patient supine, then on each side If an appreciable amount of free fluid is present, these areas will be found to shift with each change of position, the dulness always appearing in the most dependent part of the abdomen and the tympany above it. In a doubtful case, the knee-chest position can also be tried; if free fluid is present, the periumbilical region will be dull and the flanks tympanitic—the reverse of the normal picture In testing for shifting dulness, two possible sources of error must be borne in mind:

1 Loops of small or large bowel containing excessive amounts of fluid material may, when the patient changes position, shift sufficiently to alter the percussion note in a way similar to that which occurs with free fluid

2. When the fluid is due to an inflammatory or malignant process, its free flow from one part to another is often prevented by adhesions.



Fig 29 11 Correct method of testing for fluid wave Assistant's hand is exerting pressure along midline of abdomen.

Fluid Wave. This can be demonstrated with a large amount of free fluid and a tense abdomen With the patient supine, the physician places a hand on one flank and with the fingers of the other hand briskly flicks the surface of the opposite flank The impulse thus produced is transmitted through the fluid and can be felt by the palpating hand To prevent the abdominal wall from acting as a transmitting medium, an assistant must exert firm pressure with the edge of *his hand along the midline of the abdomen at the level of the examiner's two* hands Even with this precaution, particularly in a fat patient, waves may be transmitted by the wall so that in the presence of obesity this test must be interpreted with caution

Doughy Resistance. When the fluid is in pockets or its quantity is too small to produce shifting dulness or a fluid wave, it may be suspected if, on palpation or percussion, the abdominal wall presents a certain characteristic *doughy* resistance This *sign is of little practical* value *unless found during the course* of an illness such as tuberculous peritonitis, in which the development of fluid is a possibility.

X-Ray Findings

A plain film shows obliteration of the normal visceral outlines and a hazy ground-glass appearance of the entire abdomen If the fluid is under tension or

large in amount, upward displacement of the diaphragm, and perhaps compression of lung bases and upward displacement of heart, will be observed.

With a large amount of fluid or with a lesser amount if it is under tension, respiratory distress due to upward displacement and impaired movement of the diaphragm is likely. Secondary to diaphragmatic displacement, compression atelectasis of the lung bases and displacement of the heart upward and *to the left* are common Edema of the legs is usually present, it may be entirely mechanical or be partly the result of other factors associated with the underlying disease, such as low serum protein and increased capillary permeability In cases of portal or inferior vena caval obstruction, one may find distention of abdominal veins and perhaps a venous hum over collateral vessels.

Diagnostic Pitfalls

Obesity. To distinguish between true shifting dulness and fluid wave, and comparable signs due to the obesity, is extremely difficult. X-ray and other means of investigation are often necessary before the problem can be clarified

Ovarian Cyst. A huge ovarian cyst may give a picture quite similar to ascites In the latter, with the patient supine, the dulness is in the flanks and suprapubic region, and the tympany in the upper and mid-abdomen; in ovarian cyst, the intestines are forced laterally and backward, so that the dulness is anterior and the tympany in the flanks In contrast to ascites, the umbilicus does not protrude.

Acute Peritonitis

This is a suppurative process due to spread of infection to the peritoneum from a diseased intra-abdominal organ, either by its perforation or passage of bacteria through its wall without perforation Blood stream infection from a focus elsewhere is an occasional cause Depending on the dosage and virulence of the infecting agent, the patient's powers of resistance, and the efficacy of specific therapy, the process may remain localized (*local peritonitis*), or spread to involve the whole peritoneum (*general peritonitis*) The most common causes are.

1 Acute appendicitis

2 Acute cholecystitis.

3. Salpingitis or other genital tract infection in women.

4. Perforation of peptic ulcer

5 Perforation of intestinal wall from some other disturbance such as diverticulitis, cancer, ulcerative colitis, or puncture wound.

6 Necrosis of bowel wall secondary to impairment of its circulation, as in strangulated hernia or mesenteric thrombosis

7. Contamintion during a laparotomy

Local Acute Peritonitis

Since this is almost always secondary to some other intra-abdominal disturbance it is difficult, except in the advanced case, to distinguish the symptoms

and signs of peritoneal involvement from those of the underlying disorder. In fact, when a structure such as the appendix is acutely inflamed, there is probably always some involvement of its peritoneal surface. The usual manifestations are:

Pain. At onset, pain may be at the site of the underlying lesion or begin diffusely and localize later. It is variable, sometimes moderate or even mild and dull, sometimes sharp and colicky. Associated nausea and vomiting are likely.

Systemic Reaction. Anorexia, malaise, fever, and leukocytosis are the rule.

Tenderness. Usually most intense over the site of the lesion but sometimes quite general, tenderness is increased by pressure on the abdomen, an important point in differentiating peritoneal pain from colic which is often reduced by external pressure. In pelvic peritonitis it may be elicited only by rectal or vaginal palpation.

Muscular Spasm. Involuntary rigidity of the abdominal wall is usually limited to the region of the lesion. When especially severe it may cause the patient to lie with his knees drawn up, to relieve tension on the irritated muscles.

Rectal Distention. Ballooning of the rectum, detected on digital examination, is suggestive of peritoneal irritation.

Local Swelling. If a circumscribed abscess forms or segments of intestine and omentum become matted together at the site of the trouble, local swelling of the abdominal wall may be visible, or a definite mass may be felt beneath it. In *pelvic peritonitis, one can often feel, by vagina or rectum, fulness in the posterior* cul-de-sac indicative of pus or, more often, especially in gonorrheal infection, masses due to abscess formation in the Fallopian tubes.

X-Ray Findings. If an abscess forms, an indefinite mass, perhaps displacing a segment of large or small bowel, may be seen.

Subphrenic Abscess

This is a form of local peritonitis requiring separate discussion. Pus collects just beneath the diaphragm on the right side (*perihepatic subphrenic abscess*), rarely, on the left (*perisplenic*). Neither is common.

Perihepatic Subphrenic Abscess. Usually secondary to appendicitis with abscess, perforation of peptic ulcer, or acute disease of the gallbladder, this should always be suspected in any of these diseases which, despite proper treatment, continues to show the constitutional signs of retained pus such as fever, recurrent chills, and leukocytosis.

The patient complains of pain in the lower right side of the thorax and often in his right shoulder or side of the neck. Hiccough is likely. Tenderness of the ribs and *intercostal spaces overlying the liver anteriorly* and posteriorly may be found. If this is not evident *deep tenderness can often be elicited by sudden anteroposterior or lateral compression of the lower ribs*. Anteroposterior compression is effected by placing one hand over the lower ribs posteriorly, the other over the same ribs anteriorly and suddenly pressing them toward each other—lateral compression by pressing toward each other the lower lateral walls of the thoracic cage.

The lower part of the right pleural cavity may show fibrinous or serofibrin-

ous pleuritis as a result of irritation or spread of infection upward through the diaphragm. The base of the right lung may be compressed or develop signs of infiltration reflecting extension of the infection. If, as sometimes happens, the abscess cavity contains gas, the upper border of liver dulness may appear abnormally low.

Due to elevation and fixation of the diaphragm, Litten's sign is absent, a finding which is helpful in distinguishing subphrenic abscess from intrahepatic tumor or abscess, neither of which restricts diaphragmatic movement, but is not helpful in differentiating it from empyema, which also impairs diaphragmatic motion. This test has been almost entirely superseded by x-ray studies which characteristically show elevation and fixation of the right half of the diaphragm, particularly posteriorly. If gas is present, mottled subphrenic rarefaction will be seen As a rule, the pleural and/or pulmonary changes just described are seen.

Perisplenic Subphrenic Abscess. Much less common than the perihepatic type, this may follow perforation of a gastric ulcer or result from extension of infection from the pelvis upward along the left abdominal gutter Systemic evidence of continued infection, pain, and local or compressive tenderness in the flank or lower thoracic cage, occurring in a situation known to favor this complication, constitute the basis for suspecting the diagnosis In general, both local examination and x-ray studies are less helpful than in perihepatic abscess, because only a small segment of liver is on the left side so that the pus has more space in which to extend downward and thus causes less irritation and impairment



Fig 29 1
general peri
shadow not sharply defined because of [illegible] also represents secondary inflammation
Patchy density in medial portion of right lower lung field also represents secondary inflammation
On fluoroscopy, movement of right hemidiaphragm decidedly restricted.

of the diaphragm X-ray changes, if present, are comparable to those encountered on the right side.

General Acute Peritonitis

Pain. Characteristically diffuse and felt over a considerable portion of the abdomen, pain is continuous and severe but fluctuates in intensity and is marked by bouts of colic. The patient usually lies motionless, for movement of any kind increases discomfort In general peritonitis resulting from sudden rupture of the gut, pain is excruciating from the start

Nausea and Vomiting. These are present from onset As the disease progresses and intestinal paresis sets in, there is frequent vomiting or regurgitation of small amounts of brownish, foul, fecal-like material (*stercoraceous vomitus*)

Systemic Reaction. Fever is high, pulse rapid and weak, respirations elevated but not labored The patient is alert and apprehensive; his mind remains clear unless the end is near As his condition becomes more grave, his expression becomes pinched and drawn, skin cold, clammy, and of a cyanotic hue, and other indications of peripheral circulatory failure develop. Leukocyte count is high unless the infection is overwhelming, here it may be normal or low

Distention. Although initially abdominal retraction may be present, generalized distention soon appears, becoming extreme in the late stages. Tympany is pronounced Free fluid in the flanks, although sometimes said to be an important sign of peritonitis, is rarely demonstrable clinically

Tenderness. This is extreme and generally distributed over the abdomen Especially important is tenderness in the flanks in mid-axillary line appreciable on gentle palpation Its cause is peritoneal irritation at the points of peritoneal reflection, created by gravitation of infected fluid into these dependent parts

Muscular Spasm. The abdominal wall feels rigid everywhere Respiratory movements of the abdomen are diminished or absent

Diminished Peristaltic Activity. Determined by auscultation, this is an extremely important indication of general peritonitis Although it may be increased for a short time at the start, bowel activity soon becomes diminished and peristaltic sounds feeble and infrequent Later, when bowel paralysis becomes complete, the sounds disappear entirely

X-ray Findings. Paralytic ileus is indicated by increased gas in the large and small bowels and usually some increase in diameter of the latter In an upright film a fluid line may be apparent in one or more segments of small intestine The picture cannot be distinguished from that of mechanical obstruction

When peritonitis develops in an aged person or a patient worn out by chronic disease or an exhausting operation, the picture is deceptive. Systemic response and local signs, with the exception of diminished or absent peristalsis, are not pronounced

Primary Peritonitis

Usually due to streptococcus or pneumococcus, this is not infrequently seen in infants and children It is generally regarded as a blood-borne infection, al-

though the gastrointestinal tract, the transdiaphragmatic lymphatics and, in females, the genital tract have been the suspected portals of entry. This disease is a common complication of the nephrotic syndrome Onset, not infrequently preceded by severe or mild upper respiratory infection, is abrupt, with fever, abdominal pain, nausea, vomiting, often diarrhea, and severe prostration. The abdomen is distended, diffusely tender, and rigid, peristalsis is feeble or absent.

Chronic Peritonitis

This may be fibrous or serofibrous, the latter being almost always due to tuberculosis (*see below*). Fibrous peritonitis, although occasionally tuberculous, is more likely due to other low-grade infection or is the end-result of acute peritonitis. It causes trouble only when adhesions form in such a way as to hamper intestinal movement, whereupon mild gastro-intestinal symptoms or evidence of partial or total obstruction appear. If the female reproductive organs are involved, increased menstrual pain and other symptoms referable to pelvic structures are likely.

A localized form of chronic peritonitis occurs as perihepatitis or perisplenitis, the former being most likely due to pelvic inflammatory disease in the female Distinctive manifestations are missing but there may be some local pain and perhaps peritoneal crepitus audible or palpable as the liver moves with respiration.

Tuberculous Peritonitis

Tuberculous peritonitis, usually a disease of children or young adults, is caused by rupture of a mesenteric lymphnode into the peritoneal cavity, extension of disease from the intestine or a fallopian tube or, less frequently, by hematogenous seeding from a more remote focus In a doubtful case, detection of a tuberculous lesion elsewhere favors this diagnosis The process may be chiefly *fibrous* with matting together, by adhesions, of intestines, omentum, and other structures, or chiefly *exudative* with development of fibrinous exudate and considerable amounts of serofibrinous fluid

Symptoms

Onset may be insidious or sudden Fever, general malaise, loss of weight, and mild anemia are the rule. Digestive disturbances of varying degree, such as indigestion, flatulence, a feeling of distention, and constipation or diarrhea are likely, colicky pain based on partial obstruction may develop Occasionally indications of total obstruction are encountered

Signs

Distention. The abdomen enlarges because of fluid in the cavity, excess intestinal gas resulting from impaired peristalsis, or both.

Tumor-like Masses. *Scattered masses usually larger or softer than actual tumor are often felt, they are formed by loops of intestine and segments of*

omentum which have become matted together, or by collections of encapsulated fluid

Tenderness and Spasm. These are usually present but as a rule are not as pronounced as in acute general peritonitis. Peristaltic movements may be increased or decreased, depending on whether the gut is over- or underactive.

Intra-abdominal Fluid. This may vary from a quantity not detectable by ordinary clinical means to as much as is found in portal obstruction as, for example, in advanced cirrhosis of the liver As a rule, the amount lies between these two extremes It may be free within the cavity or confined by fibrous or fibrinous adhesions in pockets of varying size, the abdomen may have a certain characteristic *doughy* feel Being an exudate, it usually has a specific gravity above 1 016 and, in addition to endothelial cells and lymphocytes, contains red cells, sometimes in sufficient number to give it a bloody appearance Tubercle bacilli can almost always be recovered by culture or guinea pig inoculation, occasionally they can be found on direct microscopic examination of centrifuged sediment

X-Ray Findings

Fluid, if present in appreciable amount, creates obliteration of visceral outlines and ground glass appearance characteristic of ascites. Loops of small bowel may show fixation when palpated under the fluoroscope With a barium meal, varying degrees of kinking and obstruction of small intestine can be detected

Rheumatic Peritonitis

This term is applied to a clinical syndrome occasionally encountered in cases of active rheumatic infection The patient develops fairly suddenly acute abdominal pain often localized in the right lower quadrant, nausea and vomiting, and tenderness and spasm likewise often limited to the right lower quadrant—a picture strongly suggestive of acute peritoneal inflammation Opinion is divided as to whether there is ever a pathologic basis for the diagnosis of rheumatic peritonitis The chief importance of the clinical syndrome is that it can easily be mistaken for acute appendicitis In fact, the latter can rarely be excluded except by surgical exploration

Carcinoma of Peritoneum

Peritoneal carcinomatosis develops by direct extension or metastasis of carcinoma elsewhere, most commonly the stomach or ovary It resembles tuberculosis of the peritoneum in that there are gastrointestinal complaints, loss of weight, tumor masses and ascites However, it tends to occur in older patients, and loss of weight and anemia are more pronounced. When palpable, the tumor masses are smaller and harder than in tuberculous peritonitis but often the implantations are too small to be felt. Fever may occur but this is more likely a reflection of liver involvement

When free fluid is present, its quantity exceeds that ordinarily present in

tuberculous peritonitis. In fact, it may accumulate so rapidly as to suggest cirrhosis of the liver. It is quite similar in character to that of tuberculous peritonitis, having a relatively high specific gravity and, often, a bloody tinge. It is sometimes possible to establish diagnosis by identifying tumor cells in a Papanicolaou preparation or paraffin sections of centrifuged sediment.

Hard nodular infiltration of the umbilicus or fixed nodules on the pelvic floor are, when found, strong confirmatory evidence of cancer, especially if there are other manifestations of intra-abdominal malignant disease. The nodules in the pelvis are usually found on digital examination of the rectum and may form a definite ridge, palpable through the anterior rectal wall (*rectal shelf*) Sometimes these lesions may be felt on vaginal examination but must not be confused with the softer and less firmly fixed nodules found in endometriosis Demonstrable carcinoma elsewhere in the body is always in favor of a diagnosis of carcinoma of the peritoneum in any case showing ascites or a palpable abdominal mass. The x-ray picture is likely to resemble that of tuberculous peritonitis

DISORDERS OF THE MESENTERY

Enlarged Lymphnodes

In a thin patient, hard masses representing enlarged mesenteric or retroperitoneal lymphnodes can occasionally be felt on deep palpation Tuberculosis, lymphoma, or carcinoma is the most likely cause

Acutely inflamed nodes in the ileocecal region, the cause of which is unknown, occur not infrequently, especially in children and young adults Ordinarily they cannot be felt They give rise to symptoms and signs—particularly local pain and tenderness—often clinically indistinguishable from those of acute or recurrent appendicitis

Mesenteric Thrombosis and Embolism

Thrombosis or embolism of a mesenteric artery creates a clinical picture similar to that of acute intestinal obstruction from which it sometimes cannot be distinguished except by surgical exploration (*see* Chap 30) Onset is abrupt In the absence of an obvious cause for intestinal obstruction, thrombosis is to be suspected if the symptoms and signs appear in a patient with widespread arteriosclerosis, or embolism in one who has a potential source of peripheral embolism such as mitral stenosis with auricular fibrillation or recent myocardial infarction. Gangrene of bowel due to obstructed local circulation causes prompt rise in temperature and leukocyte count, in intestinal obstruction these appear later since impairment of blood flow to the affected loop does not occur immediately. Early appearance of blood in the stool also suggests vascular insult provided one can exclude malignant disease, intussusception and certain other causes of intestinal bleeding Some degree of paralytic ileus is observable by x-ray.

The manifestations of mesenteric venous thrombosis, which is rare, are similar to arterial thrombosis but onset and progression are slower and the prognosis somewhat better.

30

ESOPHAGUS, STOMACH, AND INTESTINE

INDICATIONS OF TROUBLE

Structural disease of the digestive system is so often simulated by some functional disturbance or by structural disease elsewhere that diagnosis of a case presenting symptoms referable to the gastrointestinal tract can rarely be reached except after thorough investigation. In recent years, x-ray and other forms of study such as gastroscopy are becoming progressively more important in this field But the growing tendency to rely entirely on them must be guarded against Other methods, particularly a carefully taken history, often contribute information without which diagnosis cannot be established The more commonly encountered indications of trouble are:

Pain. Increased tension within the alimentary tract produces a sensation of burning, fulness, or distention, or a steady ache or pain. The discomfort is usually referred to the abdominal surface directly overlying the site of the disturbance but sometimes to a distant point It may be accompanied by distress deep in the abdomen at the actual site of the lesion Pronounced increase of peristaltic activity resulting from direct irritation of the muscular coat or from effort of the gut to overcome distention or rid itself of irritating contents produces spasms of acute pain which occur at regular intervals (cramps, gripes, colic) This pain shows the same distribution as that of increased tension but is felt over a wider area It is often reduced by external pressure Irritation of the peritoneum by inflammation, stretching or pulling creates steady regional pain which is aggravated by external pressure and usually accompanied by regional tenderness and muscular spasm

The location of the pain—provided the patient can indicate it with any degree of accuracy—suggests what part of the gastro-intestinal tract is the seat of the disturbance, as shown in the following outline.

Substernal region	Esophagus
Epigastrium	Stomach, duodenum, sometimes cecum or appendix
High umbilical region	Jejunum
Low umbilical region	Ileum
McBurney's point	Cecum, appendix
Hypogastrium	Colon—except for splenic and hepatic flexures and sigmoid, which tend to refer pain to a point directly over the area involved
Suprapubic, sacral, perineal region	Rectum, sometimes sigmoid

Anorexia. If of short duration, this is usually attributable to mild gastro-intestinal upset or to a minor disturbance elsewhere, such as acute coryza. Persistent loss of

but is just as

tional state Distaste initially for only certain foods, followed by loss of appetite for all foods, is suggestive of carcinoma, especially of the stomach

Dysphagia (*see* Chap 1).

Flatulence. The sensation is one of distention and is usually described by the patient as "gas". If due to gastric or duodenal irritability, it is often accompanied by gaseous or liquid eructations, if to disease lower down, by borborygmus

Nausea and Vomiting. Although common in gastro-intestinal disorders, these are also often a prominent feature of many others toxic state such as uremia or acidosis, pregnancy, acute infectious disease, increased intracranial pressure, inner ear disease, and emotional upset Always important to determine are the relationship of vomiting to eating or to some existent pain, and the amount and character of the vomitus

Hematemesis. The vomited blood is fresh, or having remained in the stomach for some time, becomes mixed with gastric contents and looks like coffee-ground material Hematemesis must not be confused with hemoptysis If there is a sudden, spontaneous welling up of blood in the mouth, it may be difficult to decide whether it originated in the respiratory of gastro-intestinal tract If the advent of the blood is plainly associated with either coughing, vomiting or retching, the distinction is easy. Some recently ingested red-colored substance in the vomitus, such as port wine, may be mistaken for blood The two commonest causes of frank hematemesis are peptic ulcer and cirrhosis of the liver; in the latter the bleeding is due to rupture of an esophageal varix Less frequent causes are gastric carcinoma or polyp, gastritis, carcinoma of the esophagus, and rupture of an aneurysm into the esophagus Small amounts of fresh or old blood in the vomitus may be found in cases of persistent vomiting from any cause—gastritis, severe acute infection, uremia, severe acute liver disease, and certain blood dyscrasias. In disease of the mouth or respiratory passages or following operation on the nose or mouth, blood may be swallowed and then vomited Gastric carcinoma has a tendency to ooze rather than bleed profusely, and the vomitus is therefore more likely to contain coffee-ground material than frank blood When bleeding in the stomach or duodenum is not profuse, hematemesis as a rule does not occur but changed or occult blood is found in the stools.

A patient who has recently vomited blood should not be subjected to vigorous palpation of the abdomen or other zealous form of examination.

Constipation. Since the frequency with which normal persons move their bowels varies widely, constipation cannot be defined in absolute terms As many as 2–3 movements daily or as few as 1–2 a week can be regarded as normal provided that is the patient's usual pattern *Temporary* sluggishness is usually due to change of diet, water, or daily routine, or to suppression of the urge because

of haste or preoccupation. *Habitual* irregularity, the commonest form of constipation, is most likely related to improper bowel hygiene and abuse of cathartics It can also be caused by poor musculature, a tight anal sphincter, or suppression of the urge to defecate because of pain from a local disturbance such as anal fissure or acute low back strain. Much more important is *progressive decrease in frequency of movements* or need for increasingly stronger cathartics. This may ultimately reach the point where the patient can pass neither fecal material nor even gas *A definite change in bowel habits not attributable to some minor circumstance is so suggestive of a progressive obstructive lesion that it demands immediate, thorough, and repeated investigation until satisfactorily explained* Suddenly appearing intractable constipation usually indicates intestinal obstruction or paralytic ileus

Diarrhea. This is the passage of more and looser stools than is customary for the individual *Temporary* diarrhea is usually the result of some dietary disorder, mild infection, or too much cathartic Emotional upset is a frequent cause. *Persistent* diarrhea is an important symptom of structural disease of the intestine, occasionally of the stomach, but it also occurs with unrelated constitutional maladies such as deficiency states, pernicious anemia, and thyrotoxicosis Frequently it is of purely functional origin. Paradoxically, *diarrhea may be due to constipation* a mass of hard fecal material partially blocks the bowel and liquid feces find their way around or through it Failure to weigh this possibility may result in failure to detect an obstructing lesion

Tenesmus. Constant or frequently recurrent recto-anal irritability or urge to move the bowel when there is actually little material to be evacuated, often indicates disease of the sigmoid, rectum, or anal canal, but may be due to diarrhea from any cause

Melena. Blood originating in the lower ileum, colon, rectum, or anus is usually of a definite red color, that from the stomach or upper intestine is almost always altered by passage through the gut and becomes blackish or tarry, although in the case of a large amount propelled rapidly it may approach the appearance of fresh blood Small amounts from an oozing lesion high in the digestive tract, such as gastric ulcer or carcinoma, may be detected only by chemical examination of the stools. Ingestion of certain substances such as iron and bismuth give the feces a blackish hue distinguishable only chemically from that due to altered blood A red color simulating that of fresh blood can be imparted by foods such as beets and cherries

The commonest causes of fresh blood in the feces are hemorrhoids, fissure in ano, ulcerative colitis, and carcinoma or polyp of rectum or lower colon Carcinoma of the upper colon is an occasional cause, duodenal ulcer, a rare one Altered blood suggests peptic ulcer, gastric carcinoma, cirrhosis of the liver, and carcinoma of the upper colon In children, Meckel's diverticulum is one of the common causes of blood—usually fresh—in the stools *It is unsafe to attribute melena to a simple cause such as hemorrhoids until serious disease such as colitis or carcinoma of the bowel has been excluded*

Swelling. *General* enlargement of the abdomen is most likely due to obesity,

gaseous distention, pregnancy, ascites, or a large tumor or cyst. In obesity, the percussion note is normal; in gaseous distention, *hypertympanitic; in the others*, for the most part, dull or flat. Extreme gaseous distention (*tympanites*) implies intestinal obstruction or paresis, or free gas in the peritoneal cavity *Local* swelling, *if due to disease of the alimentary* canal, may be caused by tumor, inflammatory *mass, or distention*, as, for example, epigastric or left upper quadrant *prominence due to dilatation* of the stomach.

Tenderness. This usually means regional peritoneal irritation It is intensified by pressure and almost always accompanied by local muscular rigidity. *Although* pain may be referred elsewhere, the point of maximum tenderness is always over the *lesion*.

Muscular Spasm. Like tenderness, this is found immediately over the inflamed structure In nervous or apprehensive persons, both tenderness and *rigidity* must be interpreted with especial caution.

Abnormal Peristalsis. (*See* Chap 29)

THE ESOPHAGUS

Physical examination is of *little* value History and x-ray studies may establish diagnosis; esophagoscopy is frequently necessary.

Esophagitis

Acute Esophagitis. The most important causes are swallowing of some caustic or irritating substance, reflux of gastric acid through an improperly functioning cardia as in congenitally short esophagus, and prolonged intubation Foreign body will cause local inflammation and erosion Focal or diffuse inflammation may also result from extension of an inflammatory process in the mouth, appear during the course of a specific infectious disease, or occur in a bedridden or debilitated patient presumably from retention of food particles due to impaired motility, here symptoms may be masked by those of the underlying disturbance.

Substernal pain increased by swallowing and sometimes definite dysphagia are the usual complaints Vomiting or regurgitation may occur Hemorrhage or perforation is a rare complication of an ulcerative lesion. X-ray shows diffuse narrowing of the lumen, usually its lower third to half, with widening above. The mucosal folds in the affected area are thickened Fine ulceration indicated by serration or actual crater formation may be seen.

Chronic Esophagitis. Impaired function with stasis due to an intra- or extra-esophageal lesion, or persistence of acute esophagitis is the most likely cause The picture is sometimes encountered as a complication of scleroderma As a rule the symptoms are similar to those of the acute process Contraction of scar tissue may result in eventual obstruction X-ray shows changes similar to those of acute esophagitis but usually more pronounced Narrowing is greater. Following recovery from a caustic burn, total obstruction is sometimes seen

Peptic Ulcer. Usually encountered in its lower 6 cm, peptic ulcer of the esophagus may be due to secretion of acid gastric juice from an area of heterotopic gastric mucosa, or to reflux through an improperly functioning cardiac

sphincter. Pain beneath the sternum, especially after swallowing, dysphagia, and sometimes regurgitation are the rule. Hemorrhage or perforation may occur. Obstruction from cicatricial contraction is a possible end-result. By x-ray one will see evidence of esophagitis toward the lower end; an actual crater is often detected. Passage of barium is delayed by spasm and scarring. Hiatus hernia is usually present.

Benign Obstruction

The common causes of benign obstruction are:

1 Benign stricture, which may be congenital or result from cicatricial contraction secondary to previous inflammation from such causes as swallowing of lye or other caustic substance, esophagitis, peptic ulcer of esophagus, or spread of infection from outside as in mediastinal lymphnode tuberculosis Sometimes it is of unknown cause.

2 Spasm, occurring where there is a focus of irritation such as an area of ulceration

3 Achalasia, often called *cardiospasm*, a chronic neuromuscular disturbance which affects the whole esophagus. The cardia is normal The lower segment (3-6 cm) is a thin-walled, small-caliber, atrophic tube; above this the organ is enormously dilated and hypertrophied. In perhaps 15 per cent of cases, the circular muscles of the lower segment are hypertrophied and the lumen narrowed, while above, dilatation and hypertrophy, although present, are not as pronounced as in the others Symptoms at first are intermittent but gradually become continuous. This disturbance is more likely to be found in patients who are emotionally labile

4 Esophageal web, composed of strands of fibrous tissue running across the lumen and most apt to occur just below the pharyngo-esophageal junction. It is thought to be the result of previous inflammation, and is also found as a complication of the *Plummer-Vinson* syndrome.

5 Foreign body

6 Pressure from without by intrathoracic tumor or aneurysm.

Symptoms

Dysphagia. Difficulty in swallowing is the outstanding symptom of obstruction Except when due to foreign body, its onset, usually occurring in childhood or early adult life, is gradual, impairment is progressive and prolonged. At first trouble is encountered only with solid foods, later with liquids It varies in intensity from time to time, the fluctuations being often, but not necessarily, related to the degree of emotional tension The patient may complain of a "lump" beneath the sternum, but frank pain is rare unless there is associated inflammation

Regurgitation. Although common, this may not be a striking symptom, since the patient prevents it by avoiding foods which experience has taught him cannot pass the obstruction It can occur immediately after eating or later. In the long-standing case, unless the obstruction is high, the greatly dilated



FIG 30 1 Benign obstruction of esophagus
A Chronic esophagitis of lower third with dilatation above
B Achalasia of distal end with pronounced dilatation above Mottled appearance of dilated portion is due to retained food particles mixed with barium Arrows point to rugae in fundus of stomach

esophagus serves as a reservoir and large amounts of retained food may be expelled at intervals of a day or more Substernal distress, pressure, or fulness relieved by regurgitation is the rule In contrast to stomach contents, the expelled material does not contain hydrochloric acid

Indications of Malnutrition. Weakness, loss of weight, and other manifestations of impaired nutrition may appear in a long-standing case

X-Ray Findings

The character of the changes is dependent on the underlying cause Partial to complete obstruction to passage of barium is apparent; when the lesion is of long duration, dilatation above is pronounced.

Differential Diagnosis

The long course of the disease, the x-ray appearance of the lesion, and dilatation of the esophagus are the most important features serving to distinguish benign stricture, achalasia, and web from carcinoma The latter usually begins in later life, progresses more rapidly, and is of shorter duration; the patient dies before great dilatation of the esophagus can occur In a doubtful case, esopha-

goscopy, examination of withdrawn material by the Papanicolaou method, and *biopsy of tissue obtained through the esophagoscope must be performed.*

If the obstruction is due to pressure from without, onset, although gradual, is not as slow as in the cases described above. Other signs suggesting intrathoracic tumor, such as brassy cough, hoarseness, dyspnea, cyanosis, and distention of cervical veins, are apt to be found.

When a foreign body is swallowed, onset is obviously acute *Foreign body must always be considered in any patient, particularly a child, who suddenly develops substernal pain, choking and difficulty in swallowing* X-ray will usually establish diagnosis. It must be remembered that the swallowed article may not be radiopaque and hence will not be visible on a plain film In this event, obstruction can usually be demonstrated by barium study, and perhaps *the offending article outlined by a ring of barium adhering to its surface.* Sometimes esophagoscopy is necessary for diagnosis

Diverticulum

Traction Diverticulum. Strictly speaking, this is not a diverticulum but rather a distortion resulting from pull on the esophageal wall by contraction of scar tissue associated with disease of a neighboring structure, such as a tracheobronchial lymphnode tuberculosis Since the opening of the pocket is at its lower border, deglutition is not impaired, food not retained, and symptoms are absent. X-ray shows, usually near the tracheal bifurcation, a horizontal outpocketing with a wide mouth Barium is not retained.

Pulsion Diverticulum. *Herniation of mucosa through the muscular layer is* the cause It usually occurs at the pharyngo-esophageal junction but occasionally within a few cm of the diaphragm It may remain small or eventually reach huge size The lower border of the sac extends below the level of its opening, symptoms are due to consequent trapping of food The most important are progressive difficulty in swallowing, and regurgitation of small or large amounts of undigested material Paroxysmal cough and choking, especially while the patient is eating or lying down, are common, sometimes the outstanding complaint is the embarrassment caused by swishing or gurgling sounds, clearly audible to the *patient and others,* when he swallows *With time and enlargement* of the sac a serious nutritional problem may be created because most solid and semi-solid food, instead of reaching the stomach, enters the diverticulum and is later regurgitated

Physical signs are usually absent but if the diverticulum is high a small rounded mass may be found beneath the muscles in the posterior triangle of the neck, it may diminish or disappear following regurgitation

Positive *diagnosis is made by x-ray which shows a pouch usually arising from* the posterior wall of the upper esophagus and hanging downward by a narrow neck Barium enters it readily but is retained for several hours or more

Carcinoma

As in benign stricture, the initial symptom is dysphagia At the start it is often intermittent, and may be so mild as to be disregarded by the patient



Fig 30 2

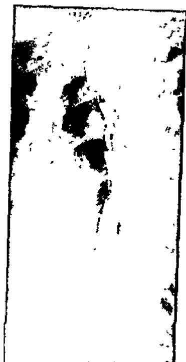


Fig 30 3

Fig 30 3. Carcinoma of esophagus Irregular filling defect in middle third

or dismissed by his physician as globus hystericus, so that obstruction may become almost complete before proper investigation is initiated Pain, which occurs late, may be. (1) substernal, midback, or cervical aching or burning, due to inflammatory ulceration or direct extension of tumor to mediastinal or para-esophageal tissues; (2) pain on swallowing due to associated spasm. Regurgitation is also a late manifestation; if ulceration has developed, the expelled material may be blood-streaked Cachexia often develops rapidly and seems out of proportion to the dietary handicap Physical signs are usually absent but *occasionally one will discover one or more enlarged metastatic* lymphnodes above a clavicle, usually the left. By x-ray one will see an irregular filling defect with destruction of mucosa and shelf-like upper and lower margins Since obstruction is usually a late development, dilatation above the lesion is not striking. Occasionally a soft tissue mass is detectable.

Esophagoscopy should be performed in any case of dysphagia which cannot

readily be explained by some demonstrable lesion such as intrathoracic tumor or a neurologic disturbance

THE STOMACH

Hiatus Hernia

Protrusion of a portion of stomach upward into the mediastinum is not uncommon in middle age or beyond, sometimes earlier. The herniated segment may vary from a small outpocketing 3–6 cm. in diameter to almost the whole of the organ. Although frequently silent, the anomaly is of importance because it can give rise to pain simulating that of angina pectoris, cholecystitis or peptic ulcer, or be a source of frank or occult bleeding. Sweet[1] divides hiatus hernia into three types:

1 Sliding type This is by far the most common. The cardiac end of the stomach extends upward through the hiatus, pushing the esophagus ahead of it This form has often been miscalled *short esophagus hernia* (*see below*) because, due to retraction and perhaps lateral deviation, the organ has appeared short although actually (as can be proved at operation) it is not With time, more and more of the stomach can work its way into the mediastinum.

2 Parahiatal type Generally called *para-esophageal hernia*, this is marked by protrusion through an independent opening *beside and close to the hiatus, not through the hiatus itself*, which remains intact The esophagus and cardia retain their usual positions but the fundus and perhaps much of the body of the stomach can work upward through the abnormal aperture. When the hernia is large, axial rotation and torsion, which are dependent on the fact that the cardia is fixed beneath the diaphragm, tend to tilt the greater curvature upward and thus create the x-ray appearance of the so-called *upside-down stomach*

3 *Infrequent variants* These are relatively rare. (*a*) *Congenitally short* esophagus with thoracic stomach The esophagus is anatomically short so that the cardia and upper part of the stomach lie within the mediastinum The intramediastinal segment assumes a cylindrical shape of nearly the same diameter as the esophagus (b) Double hernia A parahiatal hernia is accompanied by slight upward bulging of the cardia through the hiatus

Symptoms

Since it is often symptomless, hiatus hernia is likely to be incidentally discovered by x-ray *Sometimes it causes* periodic heartburn, oppression, or substernal, precordial, or epigastric pain Reference of pain to a shoulder or arm, usually the left, the interscapular region, or neck is not uncommon Another possibility is a burning or gnawing discomfort simulating that of peptic ulcer and probably attributable to associated inflammation of involved gastric or esophageal mucosa Regurgitation of small amounts of gastric contents is *common* Dysphagia for solid food, hiccough, dyspnea, and palpitation may be encountered Symptoms are more apt to appear following ingestion of a large

meal, especially if the patient lies down shortly thereafter, or increases intra-abdominal pressure by straining, bending, or lifting. As in angina pectoris, discomfort may be precipitated by exertion or emotional stress, and relieved by a nitrite, but not as consistently in any given case—that is, exertion in the same patient may at one time initiate pain, at another not. Approximately a fifth of the cases show low-grade bleeding, or, less often, frank hemorrhage from an area of gastritis in the involved portion of stomach, or from the lower esophagus if it becomes inflamed by reflux of gastric secretion secondary to impaired function of the cardia If a large segment becomes incarcerated, obstruction due to volvulus is a possibility.

Signs

Physical examination is not helpful unless a large part of the stomach is in the thorax. In this event, one may find, most likely at the left base, signs resembling those of pneumothorax or hydropneumothorax, evidence of impaired ventilation, or peristaltic sounds.

X-Ray Findings

Detectable only by a barium meal, a small hernia appears as a projection at the lower end of the esophagus; here the folds, instead of being straight, show



FIG 304 Hiatus hernia, sliding type Arrow points to circumscribed accumulation of barium in herniated portion of stomach above diaphragm, rugal folds are evident

the tortuous pattern of gastric rugae A large hernia can sometimes be recognized on plain chest films as a pocket of gas, or fluid and gas behind the heart, just to left of midline. Barium studies disclose a portion of stomach above the diaphragm.

Since hiatus hernia is so often silent, it is unsafe to blame it for any of the symptoms described above until the more serious causes, such as angina pectoris, peptic ulcer, and cholecystitis have been excluded As a matter of fact, whether to attribute the patient's pain and other complaints to a proved hernia or to some other malady, especially heart or gallbladder disease, is often a source of justifiable bewilderment to the physician The same may be said of obscure bleeding which might be due to a hiatus hernia or to some intrinsic gastrointestinal lesion such as peptic ulcer or polyp.

Gastritis

Acute Gastritis. This term is used loosely to include gastric disturbances associated with dietary indiscretion, mild food poisoning, toxic doses of drugs, acute infectious diseases, and others Excessive intake of alcohol is a common cause In its most severe form, the disease is seen in serious food poisoning, or following ingestion of some corrosive substance

The usual story is of epigastric discomfort and tenderness, thirst, nausea, vomiting, and diarrhea, their intensity varying with the severity of the trouble. In the most acute cases, epigastric pain, abdominal colic, vomiting, retching, and diarrhea are violent, especially in chemical and overwhelming food poisoning, prostration, circulatory collapse and perhaps hemorrhage are likely. Tenderness may be so marked as to suggest an acute surgical emergency but spasm is not striking As a rule, x-ray studies are not performed. If they are, excessive gastric secretion and prominent rugae will be seen

Chronic Gastritis. Until recently this diagnosis was held in ill repute because of its indiscriminate application to digestive disturbances which did not conform to any of the more readily recognizable clinical patterns, or which, by careful study, might have been proved due to peptic ulcer, gallbladder disease or, most probably, some functional disorder Recent study with the gastroscope, however, has disclosed certain changes in the mucosa which can properly be classified as chronic gastritis. Only on the basis of these findings can diagnosis be made with justification.

Symptoms and signs are not specific and x-ray appearance, though often suggestive, is rarely conclusive Digestive complaints such as anorexia, distress, flatulence, epigastric fulness or pressure, vague pain, nausea, and sometimes vomiting, are the rule The picture may resemble that of ulcer but the latter's characteristic periodicity is lacking Alkalis give less striking relief and sometimes intensify discomfort Brisk hemorrhage simulating that of ulcer sometimes occurs.

Physical examination shows no consistent findings Bad oral hygiene and coated tongue are common. Mild epigastric tenderness may be present In the hypertrophic form, thickened rugae and increased secretion will be seen on



FIG 30 5 Hypertrophic gastritis in a chronic alcoholic Note thickening of visible mucosal folds

barium x-ray studies; in the atrophic form, the contour will be smoothed out and the rugae less prominent than normal

When viewed through the gastroscope, the mucosa of hypertrophic gastritis is redder than normal and perhaps has tenacious mucus clinging to its surface, rugae are enlarged, tortuous, and between them one may see granular or warty excrescences. Areas of erosion, apt to bleed easily, may be observed In atrophic gastritis, most likely to occur in pernicious anemia or other deficiency state, the mucosa is smoothed out, pallid, and sometimes so thin that the underlying blood vessels are visible.

ULCER

Gastric ulcer occurs at almost any age but is encountered most frequently in early or mid-adult life and more commonly in males Its clinical picture is, except for certain minor differences in history (*see below*), similar to that of duodenal ulcer. Indeed, differentiation can be made with certainty only by x-ray The two are often discussed together as *peptic* ulcer but since the plan of treatment depends largely on the site of the lesion, it is wiser to consider them separately.

Symptoms

Pain is almost always the outstanding symptom. It is gnawing, localized in the epigastrium, and periodic, tending to appear 1–3 hours following each meal.

As a rule it is temporarily relieved by ingestion of food or alkali but sometimes, in contrast to duodenal ulcer, is aggravated; on occasions it will disappear spontaneously. Crampy pain suggests pyloric spasm or obstruction. Periodic vomiting also suggests one of these two complications but is not as reliable an indication for the reason that a patient without spasm or obstruction may have repeated induced or semi-induced vomiting, based on his experience that pain can be lessened by emptying his stomach. The chronic case covers a period of years and is marked by remissions and exacerbations varying from weeks to months, or years. Sometimes the first indication of trouble is hemorrhage or perforation

Signs

No distinctive physical findings are evident Epigastric tenderness is the rule but it is not much more pronounced than that found in many healthy persons. If the pylorus is obstructed (*see below*), an epigastric mass may be palpable, peristalsis seen or felt, and the signs of a dilated stomach perhaps detected However, *a palpable mass in the epigastrium should be regarded as presumptive evidence of cancer until proved otherwise*

X-Ray Findings

Repeated x-rays are essential *They* not only confirm the presence of an *ulcerating lesion, but disclose its exact site, the details of its* gross appearance,



Fig 30 6 Benign ulcer, lesser curvature of stomach Arrow indicates circumscribed projection of barium beyond gastric margin

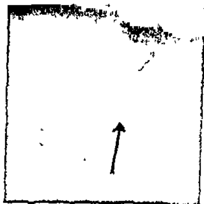


FIG 30 7 Prepyloric ulceration. A dense shadow surrounded by a ring of lesser density on posterior wall of stomach, just proximal to pyloric valve On macroscopic examination lesion was thought to be benign Microscopic diagnosis Carcinoma

and progress under treatment. The typical picture is that of a circumscribed protrusion of barium beyond the stomach wall, its edges are sharp, undermined, and, in contrast to carcinoma, do not show adjacent nodular filling defects

Analysis of gastric contents may show increased, normal, or decreased acidity The importance of this test lies in the fact that if achlorhydria is found in association with a demonstrable lesion, cancer is almost surely present The reverse is not true presence of acid does not exclude cancer.

An ulcerating lesion on the greater curvature or in the cardiac region must be considered probable carcinoma until proved otherwise by operation One elsewhere, especially if in the prepyloric segment, must also be regarded with suspicion if it fails, with proper treatment, to show rapid clinical improvement, and early disappearance by x-ray.

Gallbladder disease, kidney stone, intermittent hydronephrosis, epigastric or hiatus hernia, or a functional disorder can produce a clinical picture mistakable for ulcer.

Carcinoma

Cancer of the stomach is common beyond middle life but, contrary to popular or hiatus hernia, or a functional disorder can produce a clinical picture mistakable for ulcer.

Symptoms.

Particularly when the lesion is in the fundus, the disease is often silent until far advanced On the other hand, if it is of the ulcerative type or situated near the pylorus or cardia, symptoms are likely to appear early and, in a small proportion of cases, diagnosis can be made soon enough to provide a chance for surgical cure. The complaints vary with the nature and location of the tumor.

In carcinoma of the *fundus*, loss of appetite or distaste for certain foods previously enjoyed may be the first hint of trouble, or there may be vague complaints such as indigestion, distress or fulness after eating and, possibly, waves of nausea. In other cases, pain predominates; it may be similar to that of benign ulcer but eventually becomes constant and is then aggravated—not relieved—by food. If the lesion is in the *cardiac* region, dysphagia is prominent, if in the *pyloric region, hints of obstruction may predominate. Sometimes digestive complaints are minimal; unexplained change in bowel habits—sometimes constipation is the first symptom—loss of weight and strength, or anemia can appear first.* Constant *oozing* of blood is common with cancer, pronounced hemorrhage relatively rare, but either may be the first manifestation. In *scirrhous* cancer, because of diffuse involvement of the stomach wall without obstruction and frequently without ulceration (*linitis plastica*), virtually the whole organ can be involved before onset of symptoms. Advanced carcinoma is characterized by pronounced anorexia, great weakness, severe pain, and loss of flesh, with or without bleeding or obstruction.

Signs

As a rule, physical signs are a late development so that today, thanks to greater and earlier use of x ray and other diagnostic facilities, the disease is likely to be discovered before they become manifest. In the advanced case, unless the tumor is high and behind the ribs, a hard mass in the epigastrium is palpable and sometimes visible. It may descend with deep inspiration. In pyloric obstruction, fulness or prominence of epigastrium and left upper quadrant reflecting dilatation of the stomach may be observed, and sometimes *peristaltic waves can be seen passing from left to right. At times enlargement of liver due to metastasis is apparent and rarely, one can feel just above or below the left* clavicle, an *enlarged metastatic lymphnode (sentinel node).* Digital *examination of the rectum may reveal one or more hard nodules of tumor tissue on the pelvic floor. Additional signs of advanced carcinoma are cachexia, anemic pallor, greatly enlarged liver (often nodular) and ascites or intra-abdominal masses reflecting peritoneal or omental metastases.*

Gastric contents may show normal, diminished, or absent hydrochloric acid. Previous teaching that presence of hydrochloric acid in gastric secretion excludes carcinoma is not true. On the other hand, absence of acid in the presence of an ulcerating lesion favors the diagnosis of carcinoma, for achlorhydria is rarely found with benign ulcer. Papanicolaou study may disclose cancer cells but failure to find them does not exclude the disease.

X-Ray Findings

Barium studies show a filling defect, with absence of mucosal folds in the relief view. Localized pooling, indicative of secondary ulceration, may be evident. The involved area is rigid and fails to change in shape with peristalsis, respiratory movement, or external manipulation. By x-ray alone it is often im-

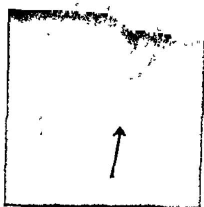


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FIG 30 8 Carcinoma of stomach Large filling defect along lesser curvature Arrow points to barium-filled area of ulceration in its center

possible to distinguish malignant from benign ulcer; features favoring the former are marginal nodularity, if present, and failure of the crater to extend beyond the stomach wall. As noted earlier, an ulcerative lesion which does not heal promptly with proper medical therapy must be regarded as possible cancer and probably treated surgically In scirrhous carcinoma, loss of elasticity is the outstanding feature, a filling defect or ulceration is less apparent.

Gastroscopic Study

This is often helpful in differentiating gastritis, ulcer, and carcinoma, especially if an adequate biopsy can be obtained However, one cannot always depend on a gastroscopic biopsy to exclude cancer, if the specimen is negative, operation may still be advisable.

Other disorders from which gastric carcinoma must be differentiated are hiatus hernia, pernicious anemia, benign tumor particularly polyp, gallbladder disease, cirrhosis of the liver, lymphoma, cancer of the pancreas and, rarely, syphilis of the stomach and intra-abdominal adhesions Any patient, especially *in middle life or beyond, who develops vague digestive complaints not explained by some simple cause must be studied with carcinoma of the stomach in mind.*

ACUTE DILATATION

This may be a complication of surgical operation, labor, injury or severe acute strain of back, other traumatic insult, or any acute toxic process In some

instances, especially following intra-abdominal surgery, it can occur independently but more likely it is associated with paralytic ileus from whatever cause (*see below*). Onset is fairly sudden with a feeling of epigastric fulness or distention, sometimes actual pain. Vomiting of large amounts of retained secretion and perhaps food sometimes occurs, but most characteristic and almost pathognomonic is frequently repeated regurgitation of small amounts of greenish, bile-stained or dark coffee-ground material.

The patient is apt to become acutely ill with rapid thready pulse, rapid shallow respiration and, often, signs of peripheral circulatory failure. If the stomach alone is involved, local examination shows epigastric and left upper quadrant fulness or prominence and, depending on whether fluid or gas predominates, dulness or increased tympany. Sometimes the enlarged organ can be outlined with a fair degree of accuracy by placing one's stethoscope receiver over the gastric region and percussing lightly toward it from various distant points on the abdominal surface: as the pleximeter finger reaches stomach margin, a definite change of note to dulness or greater tympany will be heard. Peristaltic action is diminished or absent, succussion sounds may be elicited. If function of the small or large bowel is also impaired, local signs referable to the stomach are obscured by the more widespread distention. Drainage by stomach tube establishes diagnosis and often creates great improvement in the patient's condition. X-ray, usually limited to a plain film, will show the stomach to be greatly distended with gas and fluid.

Chronic Dilatation

There are two common causes: pyloric obstruction and atony without obstruction.

Pyloric Obstruction. This is usually due to one of the following:

1. Ulcer just above or below the valve.
2. Carcinoma or, less often, some other tumor such as polyp or sarcoma.
3. Pressure from without, usually by tumor of a neighboring structure.
4. Pylorospasm secondary to ulcer of stomach or duodenum or to disease elsewhere, such as appendicitis or cholecystitis. Here the obstruction is temporary or periodic.
5. Congenital hypertrophic stenosis of the pylorus encountered in infants.

Symptoms. As a rule the patient complains of a feeling of fulness or oppression in the epigastrium, with anorexia and nausea. The characteristic pain of the underlying disorder may be present and, superimposed on it, waves of epigastric cramps secondary to overactive peristalsis. Periodic vomiting of large amounts of undigested food or frequent regurgitation of small amounts is the rule. Particles of food eaten a day or more earlier can often be identified in the vomitus. If the obstruction is prolonged, weakness, loss of weight, and other sequellae of undernourishment develop.

Signs. Epigastric or left upper quadrant fulness or prominence can usually be seen, and perhaps peristaltic waves moving from left to right, except in the obese person or when the case is so far advanced that peristalsis has become

feeble or intermittent. Succussion sounds are likely. Changes in the percussion note described above may be demonstrable A mass may be palpable in the pyloric region; although this can be due to scar tissue, as in ulcer, it must be regarded as cancer until proved otherwise.

Gastric drainage by stomach tube, and x-ray studies are important diagnostic aids As in acute dilatation, a plain film will show the stomach distended by gas and fluid. When the clinical condition of the patient permits, barium studies following deflation should be performed to determine the underlying cause.

Atony without Obstruction. Occurring in wasting diseases and also in asthenic persons, particularly those with visceroptosis, this is marked by less dilatation and less striking symptoms than pyloric obstruction Fulness or a feeling of distention, anorexia of varying degree, and perhaps vomiting are the usual complaints. Colicky pain is uncommon In a thin person, the outlines of the stomach and sometimes peristaltic waves may be seen. Succussion sounds are readily obtained. Gastric drainage yields evidence of retention x ray shows an enlarged stomach, lack of tone, and feeble or irregular peristaltic contractions

THE INTESTINES

Ulcer of Duodenum

The symptoms are similar to gastric ulcer except that the pain tends to appear somewhat later following a meal, is less likely to disappear without the aid of food or an alkali, and is rarely aggravated by food as occasionally occurs in gastric ulcer These differences are not sufficiently clear-cut to enable one to



Fig. 30 9 Ulcer of duodenum Large "clover-leaf" deformity characteristic of long-standing ulcer. Arrow points to barium-filled crater in its center, indicative of current activity.

distinguish between the two X-ray is imperative Hemorrhage and perforation are common; either may be the initial manifestation When ulcer involves the posterior duodenal wall, periodic burning or boring pain often appears in the right subscapular region or in midback at the level of the eighth dorsal vertebra, usually along with the typical epigastric pain, sometimes independently At this site, ulcer often completely destroys part of the posterior duodenal wall, involves the anterior surface of the head of the pancreas, is resistant to medical treatment, and especially prone to hemorrhage *In contrast to ulcer of the stomach, duodenal ulcer is rarely malignant*

By x-ray, acute ulcer is indicated by local pooling of barium Spasm will be evident on fluoroscopy if the deeper layers of the wall are involved, but is absent when the lesion is limited to the mucosa Chronic ulcer shows, most likely in the cap, deformity of outline secondary to scarring, spasm and pooling are dependent on the degree of activity

Acute Appendicitis

The commonest inflammatory process within the abdomen, acute appendicitis occurs at any age but most often in childhood and early adult life

Symptoms

Pain. Moderate pain, usually cramp-like, occasionally steady, is the first symptom As a rule, it starts in the umbilical or epigastric region, but soons shifts to the right lower quadrant, if the appendix becomes much distended, it may be excruciating If gangrene develops, pain will temporarily diminish or disappear, usually on the second or third day, but local signs will remain The relief experienced, by creating a false impression of improvement and hesitancy to seek medical advice, is responsible for many instances of delayed diagnosis and resultant serious consequences When perforation occurs, pain secondary to ensuing peritonitis reappears with equal or greater intensity

Nausea and Vomiting. Usually appearing within the first 24 hours following the onset of pain, these rarely persist

Bowel Disturbance. Constipation is likely at onset but diarrhea is by no means uncommon and may be responsible for a mistaken diagnosis of gastroenteritis The popular conception that diarrhea does not accompany onset of *acute appendicitis is incorrect*

Urinary Disturbance In pelvic appendicitis bladder irritation may result in urinary frequency or urethral pain at the end of micturition

Constitutional Response Fever is slight or moderate, as a rule not over 101°–102°, usually less Especially in retrocecal appendicitis, there is often none

Signs

Tenderness. Characteristically this is found at McBurney's point on superficial or sometimes only on deep palpation, but may not be elicited by either. In this event it may be brought out by pressing firmly over McBurney's point, then suddenly withdrawing the palpating hand as pressure is released, local

pain will be experienced momentarily (*rebound tenderness*). Or, firm pressure exerted on the left side of the abdomen will produce or aggravate pain on the right side (*contralateral tenderness*). In *pelvic* appendicitis, tenderness is absent in the right lower quadrant but may be found high in the right vault by rectal or vaginal palpation. Rarely, when it is sufficiently swollen, the organ itself can be felt The tenderness of *retrocecal* appendicitis is more evident in the right loin than anteriorly. Occasionally, because of failure of the cecum to descend or rotate, the abnormal position of the inflamed appendix causes the tenderness to be found in the lower midline, to the left of it, or in the right upper quadrant

Muscular Spasm. Ordinarily this is found in the region of McBurney's point but may be absent when inflammation is mild To relieve associated spasm of the psoas muscle which sometimes occurs in the severe case, the patient may lie with his right thigh and leg partially flexed, or if he stands, keep his trunk bent forward Less pronounced irritation of this muscle will be reflected as pain deep in the right lower quadrant on straight leg flexion or hyperextension. Local abdominal spasm is usually absent in retrocecal or pelvic appendicitis; in the case of aberrantly situated cecum it will be found at the same site as the tenderness

In children, old people and diabetics, the pain, tenderness, and spasm are often very slight

In uncomplicated appendicitis the leukocyte count is moderately elevated, perhaps 12,000–15,000, *but often lower and sometimes normal. In general*, a higher count suggests a complication, or some unrelated disease such as lobar pneumonia or renal infection.

Diagnostic Pitfalls

Distinction between acute appendicitis and any acute local inflammatory process in the abdomen, particularly acute salpingitis, acute cholecystitis or acute diverticulitis may be possible only at operation. Bleeding of a Graafian follicle attended, as it sometimes is, by severe sudden pain and vomiting, is also often wrongly diagnosed as acute appendicitis (*see* Chap 33). The differential diagnosis of the latter and beginning lobar pneumonia has already been discussed (*see* Chap 25) Also sometimes confused with appendicitis are acute gastro-enteritis, distention of cecum secondary to constipation, carcinoma of cecum, regional ileitis, acute pyelonephritis, ureteral stone, tubal pregnancy, myocardial infarction, and, rarely, acute pancreatitis, gastric crisis of tabes dorsalis and rheumatic peritonitis.

In a person who is more or less familiar with the symptoms of appendicitis, a vivid imagination may conjure up a set of symptoms difficult for the physician to distinguish from those of actual disease Even tenderness may be simulated but by distracting the patient's attention while palpating, one is usually able to press hard over the appendix region without eliciting complaint Apprehensive temperament and absence of fever or leukocytosis are helpful in ex-

cluding appendicitis, but in an occasional case, the picture is such that operation becomes advisable because one cannot be certain that local trouble does not exist.

The common complications of appendicitis are general peritonitis, pylephlebitis (septic thrombosis of the portal vein), and intestinal obstruction secondary to abscess or plastic peritoneal exudate

Appendiceal Abscess

Constitutional symptoms are more severe, and leukocyte count higher—usually 18,000 or more—than in uncomplicated appendicitis, a mass is often palpable in the right lower quadrant If the appendix is in the pelvis, swelling in the right lower quadrant is unlikely but rectal or pelvic examination may reveal downward bulging of the pelvic floor or a fluctuant mass in the right posterior cul-de-sac Retrocecal abscess is likely to extend upward in the loin, point posteriorly, and simulate perinephric abscess

Recurrent Appendicitis

The patient has one or more attacks suggestive of mild appendicitis, but there is insufficient basis for positive diagnosis Between episodes he is either perfectly well or has vague gastro-intestinal complaints usually with discomfort centering in the right lower quadrant Eventually, a more severe attack with characteristic indications of acute inflammation occurs

"Chronic Appendicitis"

There is rarely, if ever, clinical basis for the diagnosis of chronic appendicitis It is often incorrectly made in persons presenting vague abdominal complaints such as gas, distention, constipation, and distress, with or without pain in the right lower quadrant, and showing no conclusive evidence of other structural intra-abdominal disease Most patients in this group actually have functional disturbance of the gastro-intestinal tract or pelvic organs, usually based on visceroptosis, emotional instability, improper hygiene, and chronic fatigue A few have unrecognized peptic ulcer or intermittent hydronephrosis

In an occasional patient, symptoms which had been thought due to some chronic intra-abdominal disease such as peptic ulcer or cholecystitis disappear following operation for an attack of acute appendicitis, previously the appendix had not been under suspicion Such a case could perhaps be properly labeled chronic appendicitis but, in general, it is wiser to avoid use of this term because of the frequency with which the diagnosis leads to unnecessary surgery

Acute Intestinal Obstruction

This usually results from one of the following·

1 Strangulation of a loop in a hernial sac or beneath bands of acquired or congenital adhesions

2 Twisting of a part of a loop on itself (*volvulus*)

3 Telescoping of intestine into itself (*intussusception*).

4. Blocking of lumen by tumor, foreign body, or fecal mass
5. Contraction of an annular neoplasm or of inflammatory tissue surrounding or involving the wall.
6. Pressure from without, as by tumor of some other abdominal structure.

In the last three groups, the symptoms of acute obstruction are likely to be superimposed on those of previously existing chronic obstruction.

Symptoms

The clinical manifestations depend to a considerable extent on the cause of the obstruction and on its site. Generally speaking, the higher the lesion, the more rapidly symptoms develop, the more severe the illness, and the more swift its course. Obstruction accompanied by impairment of blood supply due to strangulation of vessels is more serious than obstruction without this complication.

Pain. Usually the first symptom, this is sudden, intense, and, in the early stage while peristalsis is active, colicky or cramp-like. It becomes a steady ache with occasional spasmodic exacerbations as the intestine distends and muscle fatigue diminishes peristalsis. If the obstruction is in the small bowel, pain is at or above the umbilicus and appears abruptly because the lesion is usually an acute insult. In large-bowel obstruction, pain is below the umbilicus unless the lesion is proximal to the hepatic flexure whereupon backing-up in the terminal ileum may center it higher. Because colonic obstruction is likely due to progressive disease such as carcinoma, prodromal pain due to partial blockage may precede that of the acute shutdown by weeks or months.

Vomiting. In small-bowel obstruction this begins almost immediately, in the large bowel, later. At first the vomitus consists of food or bile-stained fluid, subsequently, brown or blackish material which has a fecal odor is repeatedly regurgitated in small amounts.

Obstipation. Although shortly after onset, the bowel distal to the lesion may empty itself by one or two movements, there is otherwise no passage of fecal material or even gas. When the lesion is one which favors bleeding, such as carcinoma, intussusception, or diverticulitis, the evacuated material may be bloody.

Signs

Distention. If the obstruction is high, distention will be found only in the epigastrium; if the lesion involves the lower ileum general symmetrical distention is the rule and a ladder pattern may be visible (*see* Chap. 29). An obstructive process in the lower descending colon or sigmoid creates symmetrical distention but when it is higher in the colon, fulness above, and normal or collapsed bowel below give the abdomen asymmetrical contour. The wall feels tense but true spasm is not present. General tenderness of some degree is the rule.

Mass. A loop of distended intestine, a tumor, or an aggregation of inflammatory tissue is sometimes visible or palpable.

Increased Peristalsis. At first auscultation will reveal the loud high-pitched sounds of overactive peristalsis which may or may not be palpable or visible. Later, due to muscular fatigue, they become infrequent or disappear entirely.

Systemic Response. Initially the patient does not appear seriously ill but within a few hours following onset of high obstruction, or a somewhat longer interval following low obstruction, the signs of shock appear. The expression becomes anxious and drawn, tongue dry and parched, skin cold and clammy, color slightly cyanotic, pulse rapid and thin, and blood pressure depressed. Fever and leukocytosis appear when local circulation is impaired and gangrene of bowel develops, but temperature falls as circulatory failure increases

X-Ray Findings

Whenever obstruction is suspected a scout film of the abdomen and a barium enema should always precede study by barium meal The latter procedure should not be performed unless absolutely necessary because the barium mass is capable of clogging the gut at the site of the lesion and converting partial into complete obstruction

In *small bowel* obstruction, one or more loops are distended by gas, or gas and fluid An upright film is often required to demonstrate a fluid level, not evident in the supine view In low obstruction, dilatation of numerous loops



FIG 30 10 Dilated, gas filled loops of small intestine in a case of obstruction by a band of adhesions

above the lesion may create the occasionally observed ladder pattern. When the *large bowel* is blocked, pronounced dilatation is seen above the lesion If acute shut-down is superimposed on a long-standing lesion, secondary dilatation of small bowel may also be evident.

Diagnostic Pitfalls

Acute obstruction must be differentiated from other acute insults within the abdomen *such as cholecystitis, pancreatitis*, and, especially, acute intestinal perforation and mesenteric thrombosis or embolism, in all of which severe pain, vomiting, and distention are prominent In these cases, however, one is apt to find more local tenderness, more systemic evidence of infection and higher leukocytosis, but less striking peristaltic activity The history is always important. On physical signs alone it is impossible in the early stages, to distinguish acute intestinal obstruction from general peritonitis In the latter, fever and tenderness are prominent and diminution or disappearance of peristalsis occurs early Here, too, history is of the utmost importance.

Paralytic Ileus

Passage of intestinal contents is stopped by paralysis of a segment of bowel. A mild form probably accounts for the constipation and slight distention often seen at the onset of such acute intra-abdominal diseases as acute appendicitis and cholecystitis In its most severe form it is encountered in acute general peritonitis, mesenteric thrombosis, severe systemic infection such as pneumonia, typhoid fever, or ulcerative colitis, some cases of acute spinal cord disease, perhaps in fracture of the spine or acute back strain, and following operations, especially those within the abdomen.

Although rapid, onset is not as abrupt as in acute mechanical obstruction The abdomen becomes distended, feces and gas fail to be passed by rectum, and frank vomiting or, more likely, repeated regurgitation sets in Peristaltic activity, best determined by auscultation, is greatly diminished or absent The patient becomes acutely ill, signs of peripheral circulatory failure may develop The plain abdominal film is similar to that of mechanical obstruction. The two disturbances can rarely be differentiated by x-ray, although a lesser degree of distention favors paralytic ileus

It is difficult to distinguish between paralytic ileus and ileus appearing as a feature of acute general peritonitis In the former one finds early elevation of pulse rate out of proportion to the rise in temperature, and peristaltic activity diminished or absent from the start. In spreading peritonitis, pulse and temperature charts rise together, and diminution of peristaltic sounds occurs later.

Chronic Intestinal Obstruction

The most common causes of chronic obstruction are tumor in bowel wall, especially carcinoma of the colon, scar tissue contraction in inflammatory disease such as ulcerative colitis or regional ileitis, and fixation due to invasion by some intra-abdominal malignant process Fibrous adhesions occasionally cause pro-

gressive obstruction, usually of the small intestine, but not as commonly as is popularly supposed. This situation is most likely to be found in patients with a history of previous operation *or intra-abdominal infection. Any chronic* obstruction, if untreated, may eventually reach the point of acute shut-down.

Symptoms

When the obstruction is due to an active inflammatory disease such as ulcerative colitis, the picture is colored by symptoms such as cramps and diarrhea attributable to the underlying malady, in other cases, the lesion may be well-advanced before there are any indications of trouble.

Pain. The *usual story is* one of intermittent, vague distress in mid- or low-abdomen, perhaps relieved by bowel movement or passage of gas Later discomfort increases and bouts of colicky pain develop

Bowel Disturbance. Constipation *always* appears *early and may be the first* symptom It may be continuous or periodic but is always progressive; a history of need for more frequent and stronger cathartics is usually obtainable If the lesion is at or beyond the hepatic flexure, diarrhea may alternate with constipation and be so pronounced as to conceal it, this is also true of the active inflammatory disease Blood in the stools is common and may lead to a carelessly made diagnosis of hemorrhoids This is particularly true of carcinoma of the rectum and colon

Signs

Distention. This appears gradually. At first it is perhaps localized just above *the site of the obstruction but becomes more general as gas and other intestinal* contents accumulate higher up

Increased Peristalsis. When the small bowel is obstructed, peristaltic waves *can be seen and felt and the typical sounds of increased activity heard (see* Chap 29) Visible peristalsis alone is not significant since, especially in a thin person, peristaltic waves may be seen in the absence of trouble To be certain, one must feel the alternate hardening and softening as the waves pass, or hear the sounds of overactivity Increased contractions of a segment of large bowel appear as alternate rising and falling with associated hardening and softening of overlying abdominal wall Increased sounds are the rule. In a long-standing case, fatigue of muscles will result in greater distention but lessened activity.

Mass One may feel through the abdominal wall or by rectum or vagina, tumor, inflammatory tissue, a loop of distended intestine, infiltrated or inflamed mesentery or omentum, or a collection of hard fecal material (or barium from previous x-ray examination) impacted above the obstruction If, as often happens, the patient has taken repeated doses of mineral oil in an effort to overcome the constipation, his abdomen above the lesion may have a doughy, putty-like feel due to accumulation of fecal material mixed with oil.

X-Ray Findings

A plain film of the abdomen shows a *picture indistinguishable* from acute obstruction To locate the site and probable cause, barium studies are ncessary.

Again it must be emphasized that in suspected obstruction, barium enema should always precede barium meal; the latter must be given with caution and only when regarded as essential.

Carcinoma of Large Bowel

Generally supposed, like cancer elsewhere, to occur chiefly in later life, carcinoma of the large bowel is, in fact, by no means limited to older people; it is relatively common in early adult life and even in youth. Blood in the stools is often the earliest symptom but may not appear until late Otherwise, especially when the transverse or left colon is the site of the lesion, the picture is that of chronic progressive obstruction Anemia not attributable to bleeding, with associated weakness and fatigue, is often the first indication of cancer of the cecum or ascending loop, here, barring ileocecal valve involvement, obstruction is a late manifestation because of the relatively large lumen. Occasionally a mass will be discovered in the absence of symptoms In the far advanced case, cachexia, anemia, and other systemic manifestations of malignant disease appear, but acute obstruction may precede them.

By barium enema, carcinoma on the left side, or in the transverse colon, usually appears as an annular filling defect with loss of mucosal markings, some



Fig 30 11 Filling defect of ascending colon due to carcinoma

degree of obstruction, and perhaps ulceration. A cauliflower-like intraluminal mass usually arising from one wall is characteristic of a process originating in a

abdominal cramps or distress, especially if there is any change in bowel habits, blood appears in the stools, or unexplained anemia is present.

Diverticulosis and Diverticulitis

Diverticulosis. Diverticula are pouch like herniations of intestinal mucosa through the muscular wall. They can occur singly or multiply anywhere in the large bowel, most commonly in the descending loop or sigmoid Occasionally they are found singly or in small numbers in the duodenum or small intestine. They often persist for years without giving rise to any symptoms (*diverticulosis*), but a diverticulum is capable of becoming inflamed and creating symptoms at any time (*diverticulitis*).

Acute Diverticulitis. With the exception of Meckel's diverticulitis (*see below*), or the rare case in which a duodenal diverticulum is thought to create symptoms simulating ulcer or stasis, diverticulitis is virtually confined to the colon. One finds a picture similar to that of acute appendicitis except that the pain, tenderness and spasm are more likely to be found on the left side. Urinary frequency or other indication of bladder involvement may be present if the lesion is in contact with the bladder wall Perforation with local abscess formation or, rarely, diffuse peritonitis may occur.

By barium enema a diverticulum appears as a well-circumscribed barium-filled pocket extending beyond the bowel wall; it may retain the medium for several days after evacuation When acute inflammation supervenes, fluoroscopy will reveal spasm and local tenderness over the involved segment. Secondary abscess may create an extrinsic pressure defect and occasionally barium will be seen extending through the wall into the abscess cavity.

Chronic Diverticulitis. A slowly progressive inflammatory process results in marked local thickening of bowel wall, often sufficient to create partial or total obstruction Superimposed attacks of acute diverticulitis may occur. A mass may be palpable; sometimes it changes size or tends to appear and disappear from day to day Gross bleeding, perforation, and development of a fistulous communication with the bladder are possible complications X-ray shows diffuse narrowing with one or more diverticulous outpocketings of the involved segment. Spasm, tenderness, and abscess formation are absent unless an acute episode supervenes.

It is often impossible to distinguish between chronic diverticulitis and carcinoma except by surgical exploration The two may coexist X-ray study may be helpful· in simple diverticulitis mucosal markings in the affected segment are usually visible; in carcinoma, absent.

Meckel's Diverticulitis. Meckel's diverticulum, due to persistent patency of



FIG 30 12 Diverticulosis of transverse colon

A Barium enema Bulb-like pouches filled with barium or a mixture of barium and fecal material extend beyond normal borders of bowel Narrowing of lumen and prominence of haustrations are due to spasm

B. Film taken following evacuation of enema Diverticula remain outlined by barium

the intestinal end of the vitelline duct, is a small pouch branching off the ileum about 80 cm above the ileocecal valve. Acute inflammation of this anomaly is often mistaken for acute appendicitis but the pain, tenderness, and spasm are found near the umbilicus, usually just to its left Recurrent oozing or gross hemorrhage may occur Either is usually accompanied by the indications just mentioned Partial or total obstruction can result from persistent low-grade inflammation. X-ray is rarely as helpful as one would like Occasionally, following a barium meal, the pouch will fill, or localized narrowing of the ileum can be detected.



Especially in a young person, this anomaly must always be considered as the possible site of unexplained gastrointestinal bleeding.

POLYP

One or more polyps may occur in the stomach or anywhere in the intestine; the sigmoid and rectum are favored. A polyp will vary from a small excrescence to a quite large sessile or pedunculated mass. As a rule, it is an adenomatous process. Multiple polyposis of the colon has a tendency to occur in families. Polyps may exist for years without symptoms or create attacks of melena, diarrhea, vague abdominal distress, or actual cramps. A lesion in the rectum or sigmoid can cause painful or difficult defecation, or a sensation of incomplete evacuation. Gross bleeding from mechanical block, intussusception, and change to carcinoma are important hazards. On a mucosal relief film, polyp of the stomach will be indicated by derangement of the mucosal pattern, and a circumscribed intraluminal filling defect. A lesion in the colon may show a circumscribed defect in the barium column, on the postevacuation film, one should see interruption of the mucosal relief pattern by a rounded, circumscribed intraluminal defect, and failure of bowel to collapse at this point. Double contrast films may reveal a polyp not detected by conventional x-ray studies.

DIFFUSE INFLAMMATORY DISEASES OF LARGE BOWEL

Amebic dysentery, ulcerative colitis, and bacillary dysentery are all characterized by inflammatory and destructive changes in the large bowel. When a case is seen early, it is often possible to distinguish between the three on the

basis of clinical, laboratory, x-ray and sigmoidoscopic studies. But in some instances, especially in the late stages, one cannot be certain of the nature of the process Any one of them may occur in an acute, fulminating form with severe systemic reaction, diarrhea, abdominal cramps, tenesmus, and mucus, blood and pus in the stools. Or the course may be chronic, with exacerbations of mild or moderate severity alternating with periods of relatively good health, in general, the trend is progressively downward. During an exacerbation one finds indigestion, diarrhea sometimes alternating with periods of constipation, abdominal discomfort, and cramps. Physical examination shows diffuse or local tenderness, perhaps distention and at times indications of hyper- or hypoperistalsis, but there are no specific signs Between times, the patient may be entirely well or be bothered by the same symptoms in a mild form The extent to which the stools contain abnormal elements varies with the acuteness of the process Without proper treatment, death may eventually occur from malnutrition, toxemia, persistent hemorrhage, general peritonitis with or without perforation or, especially in ulcerative colitis, superimposed carcinoma

Amebiasis. One of the pictures described above is often encountered but it must be emphasized that many cases of this disease are virtually asymptomatic These represent an especial health hazard from the carrier standpoint. Although it can occur in any part of the large bowel, amebiasis is most likely to appear in the cecum or rectosigmoid, at the former site an acutely initiated attack may be confused with acute appendicitis In the long-standing low-grade case, a granulomatous lesion (*ameboma*) may appear anywhere in the large bowel, most likely the cecum It is sometimes large enough to be palpable. Failure to exclude amebiasis as the cause of a local lesion often leads to the incorrect diagnosis of carcinoma

Barium x-ray studies show either no appreciable change, or multiple small ulcerations in any segment, usually cecum or rectosigmoid When the process is chronic and fully established, the affected segments are profoundly altered, showing extensive ulceration, contraction, gross thickening and perhaps filling defects of the ring type Such a picture cannot be distinguished from those of other inflammatory or malignant processes In an occasional case, symmetrical or asymmetrical contraction of the cecum is readily confusable with that due to carcinoma or tuberculosis.

The sigmoidoscopic appearance of the bowel differs to a certain extent in the three inflammatory diseases Visible changes may be differentiated by an experienced observer but a definite diagnosis always requires examination of stools for amebae and in early cases, cultures for the bacillary dysentery group of organisms When early amebiasis involves the rectum or sigmoid, the mucous membrane appears slightly irritated with scattered, discrete shallow ulcers, usually larger than those noted in non-specific ulcerative colitis. Occasionally it seems normal In the well-established chronic case, one may see definite punched-out ulcers filled with exudate, averaging 2–4 mm. in diameter but often much larger; inflammatory reaction around them is slight, the appearance being that of indurated ulceration with infiltration Motile amebae can

almost always be found in material obtained from a crater by careful wiping or curetting. The discovery of the organisms, the discreteness of the lesion, and the relatively normal appearance of the mucous membrane as a whole are the important diagnostic features.

When amebiasis is suspected, when vague gastro-intestinal complaints, especially diarrhea, cannot be readily explained, and sometimes in a case thought to be carcinoma, direct examination for cysts and active amebae in stools and material obtained from a lesion through the sigmoidoscope must be performed. In well-trained hands, cultures are also valuable Since *E. histolytica* is not consistently passed in the stools, multiple examinations may be required. In some cases, especially if adequate laboratory facilities are not available, a course of anti-amebic therapy is indicated to exclude the disease.

Acute amebic hepatitis, with or without abscess formation, is a common complication. It may occur during an attack of dysentery, months or weeks after such an attack, or, in a fair proportion of cases, without a history suggesting previous amebic disease of the intestine The liver abscess may rupture or penetrate into the pleural cavity or lung, causing the signs of pleuropulmonary suppuration Occasionally lung abscess arises from blood stream transmission of the organisms (*see* Chap. 31)

Ulcerative Colitis. The cause has never been determined Some observers believe that emotional factors are totally at fault, many others regard them as contributory. Still others believe that an appreciable number of cases represent the end-result of previously unrecognized acute bacillary dysentery The disease may affect part or all of the large bowel, rarely it extends upward into the terminal ileum. The rectosigmoid is most frequently involved, the process, as a rule, starting here and spreading upward as time goes on Onset may be *gradual or sudden,* with symptoms and signs of varying degree. *In the acute* fulminating case, fever and other indications of a severe toxic state are present, discharge of gas, blood, pus, or mucus may be almost constant. Often these manifestations appear more gradually and with less intensity. In a small percentage of cases, the disease begins in the cecum and tends to spread downward. When only the proximal colon is involved, systemic signs are likely to overshadow the diarrhea and other bowel symptoms In the chronic case, remissions and exacerbations are the rule but the patient is rarely entirely symptom-free and stools are rarely normal *Abdominal discomfort and lesser attacks* of diarrhea are virtually continuous In any phase, tenesmus, anal irritation, and perianal inflammation with perhaps abscess and sinus formation are *likely*

Barium x-ray studies in the early stages show spasm, slight reduction of haustrations, and superficial ulcerations indicated by serration of the margins and fleck-like accumulations of barium in the mucosal folds Later, one observes extensive involvement of the colon, with shortening, absence of haustrations, smoothing out of borders, loss of mucosal markings, and narrowing of lumen (*lead-pipe colon*), pseudopolyps, actually islands of mucosal hyperplasia between areas of destruction, may appear as circular, irregularly placed filling



Fig 30 14 Ulcerative colitis, relatively early Reduction of haustrations Granular appearance of air-containing segment of descending colon is due to ulcerations Marginal serration visible on original film has been largely lost in reproduction



Fig 30 15 Ulcerative colitis, early Spot film of barium-filled sigmoid showing typical marginal serration



FIG 30 16 Chronic ulcerative colitis, advanced Barium enema showing narrowing of lumen, loss of haustrations, and some shortening of colon.

defects At times, the terminal ileum appears rigid, dilated and without normal markings but this is thought, as a rule, to be edema and not true ulcerative disease

On sigmoidoscopy in the early stages, the mucous membrane from the internal sphincter upward presents signs of diffuse inflammation, with injection, excessive mucous secretion and, on wiping, numerous points of capillary bleeding Somewhat later one sees superficial miliary abscesses diffusely scattered throughout the entire mucous membrane, and appearing as pinhead, yellowish-gray spots When rubbed gently with a sponge, yellow exudate is removed and slight depressions which bleed readily are noted. The mucosa is obviously thickened, edematous and has lost its sheen, the lumen is narrowed In the [illegible]

[illegible] irregular shape may be observed, these are larger than the original ones and show obvious infiltration at the level of the mucous membrane. Scattered

pseudopolyps 2–4 mm in diameter are likely. The lumen is often greatly constricted.

In addition to local complications such as perforation, obstruction, perianal or rectal abscess or fistula, and cancer, one may encounter acute arthritis, erythema nodosum, tendency to thrombophlebitis and various manifestations of impaired nutrition. Portal cirrhosis is likely in long-standing cases. The frequency with which carcinoma of the large bowel develops in chronic ulcerative colitis must always be kept in mind.

Bacillary Dysentery. This is likely to appear in epidemics The manifestations vary from mild diarrhea with slight fever to those of acute fulminating disease with sudden onset, high fever, cramps, diarrhea, tenesmus and bloody, mucopurulent movements The x-ray and sigmoidoscopic pictures cannot be distinguished from those of acute ulcerative colitis although in dysentery one may observe more diffuse purulent inflammation than in ulcerative colitis. Positive diagnosis can be made only by recovering the organism on stool culture, a procedure which, as a rule, is reliable only during the first week or so.

With proper treatment most patients recover in a short time. The neglected case may develop into a prolonged chronic illness resembling chronic ulcerative colitis from which it cannot be distinguished radiologically or sigmoidoscopically Serologic studies may be tried but are of questionable value.

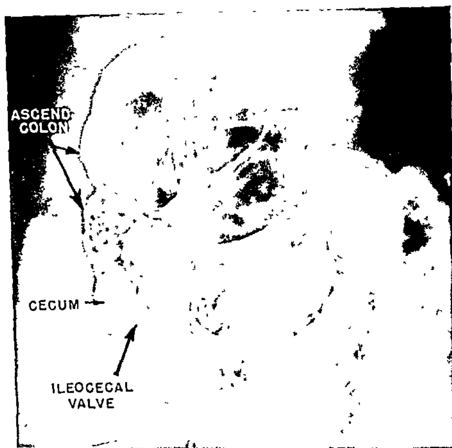
Uremic Colitis. Acute hemorrhagic inflammation of the alimentary tract, particularly the colon, is frequently found in terminal uremia Bloody diarrhea or, occasionally, frank hemorrhage, may occur but often there are no local manifestations and the complication is discovered *post mortem*

Intestinal Tuberculosis

In this country, the incidence has diminished greatly during the past two decades, probably because of a combination of better control measures, earlier recognition of pulmonary tuberculosis, and its treatment with specific agents As a rule, intestinal infection is a complication of active pulmonary disease and parallels its severity, being much more frequent in advanced than in minimal cases The cecum, terminal ileum, and ascending colon are the favored parts. Except in the advanced stage the lower colon is free of trouble, the reverse of the usual picture in ulcerative colitis

The patient may be entirely asymptomatic or have mild complaints, such as loss of weight, fatigability, low-grade fever and non-specific digestive disturbances. Intestinal involvement is usually discovered by barium x-ray studies prompted by digestive symptoms, or performed as a routine measure in a patient with known pulmonary tuberculosis. In advanced intestinal disease, one will find abdominal cramps, tenesmus, diarrhea with bloody, mucopurulent stools, general abdominal tenderness, and high fever. A mass, representing a granulomatous process or matted omentum and intestinal loops, may be palpable.

Barium studies in the early stages may show nothing more than bowel irritability. Later, one will observe ulceration, constriction, segmentation or per-



irregularity of

haps granulomatous masses with filling defects, involving the lower ileum, cecum, or ascending colon. Rapid passage of barium through the terminal *ileum and cecum is the rule. In the small bowel, the picture may be mistaken* for regional ileitis or sprue, in the cecum or ascending colon, for carcinoma or ameboma. Occasionally such widespread acute involvement of the lower as well as the upper colon is created by tuberculosis that one cannot distinguish it radiologically from ulcerative colitis. During the course of specific therapy, multiple constrictions are prominent, they rarely proceed to total obstruction and eventually disappear.

Sigmoidoscopic examination is frequently negative since the process is restricted to the proximal colon. If the disease reaches the sigmoid or rectum one may see, tending to circle the bowel, one or more isolated ulcers which are large, irregular, shaggy, and have undermined edges, partial healing may narrow the lumen.

In a doubtful case, the presence of active pulmonary disease provides strong

presumptive evidence in favor of intestinal tuberculosis. Detection of tubercle bacilli in the stools is not as helpful as one would like because, in the open case of pulmonary disease, organisms reach the bowel as a result of swallowing of infected secretions.

Primary intestinal tuberculosis, regarded as a bovine infection, is currently rare in this country. It tends to be more hyperplastic than ulcerative and have less pronounced constitutional and local manifestations. A palpable mass is likely. Clinically and roentgenologically, this form is especially likely to be mistaken for cancer or ameboma, and may require surgical exploration for positive diagnosis

Regional Enteritis

Also known as *regional ileitis*, this is a chronic granulomatous disease of the small bowel, most often encountered in young adults. Although usually limited to the terminal ileum, in some instances it extends into the cecum and proxi-



Fig 30 18 Advanced regional enteritis Barium meal A Loops of normal small intestine proximal to diseased segment B Distended, partially involved loops of ileum separated by masses of inflammatory tissue C. Distal portion of ileum markedly narrowed evidence of long-standing disease. D Normal cecum.

mal colon. Occasionally the process will crop up in additional segments of small intestine; between them lie healthy segments. Rarely, the small bowel is completely involved. The disease is progressive, tends to proceed to partial obstruction, ulceration, abscess formation, and development of fistulas. Onset is sometimes acute with a picture readily confused with acute appendicitis; in fact, the diagnosis is sometimes made at operation for suspected appendicitis. As a rule, the earliest symptom is *gradual development of persistent diarrhea* with mucus, but rarely blood, in the stools. Indications of low-grade infection—moderate fever, leukocytosis, and perhaps loss of weight—are present. Later one finds greater systemic response, poor nutritional state, alternating constipation and diarrhea, and pain, tenderness and perhaps a mass in the right lower quadrant

In the early stages, barium studies usually show hypermotility. Later a section of *small bowel shows narrowing of its lumen, rigidity, absence of normal* mucosal markings with, sometimes, dilatation immediately proximal to the diseased area Multiple segments may show these changes but between them sections of normal appearance will be found Extension into the cecum will be indicated by the serrations of fine ulceration, and usually some contraction. Occasionally, similar changes will be noted further down. Obviously the sigmoidoscopic findings are negative

FUNCTIONAL GASTROINTESTINAL DISORDERS

Symptoms ordinarily associated with gastrointestinal disease are encountered in many patients in whom no structural systemic or local disease—gastrointestinal or otherwise—can be demonstrated The patient may have acute attacks of abdominal discomfort, with diarrhea, constipation, or both, lasting a few days and relieved by rest and regulation of diet Chronic cases, which are even more common, show wide variation of symptomatology. The typical picture is that of poor digestion, distress following meals, eructations of gas or fluid, nausea, sometimes vomiting, constipation or diarrhea or both, and pain referable to, and often shifting from, one part of the abdomen to another General manifestations such as headache, nervousness, easy fatigability, weakness, poor appetite, and loss of weight are characteristic. There may be a low backache, excessive menstrual pain, and other complaints unrelated to the digestive apparatus. Such persons almost always show emotional instability, indicated by nervousness, undue sensitiveness, tearfulness, anxiety and such signs of vasomotor instability as increased sweating—especially of hands and axillae—cold extremities, and dermatographia Symptoms become intensified during periods of *emotional stress or physical or mental fatigue*

For some unexplained reason, this symptom complex is more likely to be found in persons with the body habitus known as *visceroptosis*, characterized by a thin, narrow type of build with an acute costal angle, exaggerated curves of the back, low position of thoracic and abdominal organs with palpable liver edge and right kidney, and perhaps tender and palpable cecum and sigmoid By x-ray one will see a long, low heart, long "fish-hook" stomach,

low cecum, "hammock" type transverse colon, and long, low, redundant sigmoid. The conception that visceroptosis (or gastroptosis, the condition popularly and erroneously known as "fallen stomach") is the actual cause of the symptoms is probably incorrect, for many persons with this variant are perfectly healthy and happy A more plausible explanation is that, although the function of the digestive organs may to some extent be handicapped by low position, other factors, particularly fatigue and emotional instability, are largely responsible for the complaints

The frequency with which a functional gastro-intestinal disorder is mistaken for structural disease and needless surgery is performed makes recognition of this syndrome of great importance. The diagnosis depends on a carefully taken history, proper evaluation of the patient's emotional status and behavior, and exclusion of structural disease by the usual methods of investigation

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LIVER, GALLBLADDER, PANCREAS, AND SPLEEN

LIVER

Since the functions of the liver can be maintained by only a small amount of normal parenchyma, almost all of the organ may be diseased before clinical evidence appears. Depending on its nature and extent, liver impairment will, as a rule, create some of the following manifestations:

Pain. Pain referable to the liver presumably arises from stretching of the capsule due to parenchymal swelling, as in passive congestion or hepatitis, or its involvement by an inflammatory or neoplastic process. Characteristically, it takes the form of steady, right upper quadrant or epigastric ache; when the diaphragmatic surface is involved, it may be referred to the right shoulder. With acute congestion or great enlargement, a sense of heaviness is described. For reasons not understood, bouts of sharp right upper quadrant pain resembling biliary colic (*see below*) often occur in cirrhosis and severe hepatitis.

Gastro-Intestinal Disturbances. Anorexia, nausea, and vomiting are common in many forms of liver disease.

Fatigue. Progressive fatigability and loss of strength are important symptoms in all types of parenchymal liver disease.

Loss of Flesh. This is most pronounced in severe acute processes, malignant disease, and late cirrhosis. Most striking in the face and extremities, the loss may not be evident on the weight record because of the counterbalancing effect of coexistent edema and ascites.

Neurologic Disturbances. Depression is common in the presence of jaundice. In severe liver insufficiency, headache, apathy, disorientation or other personality change, drowsiness, tremulousness, and eventually delirium, convulsions, and coma are the rule. Irregular flapping movements of the outstretched extremities (*see* Chap. 6), grasp reflexes, extensor or plantar responses, or rigidity of the trunk or limbs may be encountered. Coma is sometimes precipitated by the injudicious use of certain narcotics or sedatives, especially opium derivatives and barbiturates; what would ordinarily be a normal dose becomes a toxic one because of inability of the liver to detoxify the drug.

Fever. Low-grade fever is encountered in hepatitis and cirrhosis. It may be moderately high in severe acute hepatitis or carcinoma; in fact, malignant

disease of the liver is a common cause of obscure fever. Septic chart with chills, sweats, and leukocytosis is present in *septic processes such as acute suppurative* cholangitis and liver abscess, and sometimes in metastatic carcinoma.

Jaundice. *(See below.)*

Tenderness. This is found in the right hypochondrium or epigastrium. When *moderate, it is most frequently due to congestion,* acute or subacute hepatitis, or early cirrhosis, when marked, to a suppurative process, especially one which reaches the hepatic surface With a deep-seated abscess, discomfort may be elicited only by the compression method of palpation Although occasionally encountered, tenderness, as a rule, is not a striking feature of malignant disease.

Enlargement of Liver. *(See below.)*

Diminution in Size of Liver. Occurring in severe hepatitis and the late stages of postnecrotic and cardiac cirrhosis, this is occasionally detected by x-ray, rarely, by physical examination In severe hepatitis, shrinkage indicative of progressive disease can sometimes be established by day-to-day percussion, one finds gradual recession of the upper and lower borders (*see* Chap. 29)

Edema and Ascites. In hepatitis and cirrhosis these may be due to disturbance of plasma protein production, interference with local venous or lymphatic circulation, increased capillary permeability, or combinations of these and other possible causes If ascites appears without disturbance of the plasma proteins, a malignant process is more likely than diffuse intrahepatic disease

Signs of Portal Obstruction.

1. Slight to moderate enlargement of spleen Determination of splenic size is important in differentiating between jaundice due to hepatic disease and to biliary tract obstruction by stone or tumor. enlargement is present with the former, rarely with the latter unless there is secondary biliary cirrhosis

2. Dilated esophageal veins (*esophageal varices*). These are detectable by barium x-ray studies or esophagoscopy By x-ray they appear as tortuous, linear or polypiform filling defects usually confined to the lower third of the esophagus but sometimes extending higher Occasionally similar defects are evident in the upper part of the stomach

3 Hematemesis from ruptured esophageal varix

4 Melena from ruptured esophageal varix or hemorrhoidal vein.

5. Ascites

6. Dilatation of abdominal veins (*see* Chap 29)

7 Dilatation of hemorrhoidal veins

Blood Disorders. In acute or chronic parenchymatous disease, bleeding tendency is common and often pronounced, most likely from impairment of prothrombin synthesis, rarely, from thrombocytopenia Presumably nutritional, anemia, usually normocytic, normochromic but sometimes macrocytic, is the rule in the chronic case, a hemolytic element may be a contributing factor in advanced cirrhosis The blood picture *may be confused* by acutal blood loss.

Spider Angiomas. Frequently observed in liver insufficiency, especially that associated with alcoholic or postnecrotic cirrhosis or chronic hepatitis, rarely,

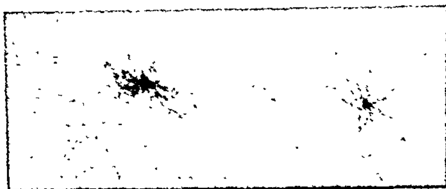


FIG 31 1 Spider angiomas on upper back in a case of alcoholic cirrhosis of liver

in severe acute hepatitis, these are regarded as an important indication of parenchymatous as opposed to other forms of liver disease They appear quite suddenly, especially on the face, arms, neck, and thorax. *When limited to the face*, they are likely to be of less significance, since here they are occasionally observed without known cause. They sometimes *occur without disease during* normal pregnancy, disappearing after parturition.

A spider angioma is an irregular, roughly circular 2–5 mm, red to bluish-red, flat to slightly elevated patch of discoloration from which dilated tortuous capillaries or venules radiate outward in dendron-like fashion for a distance of 0 5–1 cm The central point probably represents an arteriolovenular aneurysm. By pressing a microscope slide lightly over the lesion, one may observe alternate flushing and fading synchronous with arterial pulsation; firm pressure will obliterate the discoloration Occasionally pulsation can be felt Spider angiomas must be distinguished from small patches of dilated venules which lack the spider-like conformity. Clinically unimportant, these are often found along the lower rib margins in normal individuals and on the face in alcoholics and persons repeatedly exposed to the elements

Liver Palms. Mottled redness of the thenar and hypothenar eminences, occasionally of the fingers and toes, is often seen in liver insufficiency secondary to chronic parenchymatous disease This phenomenon is also encountered in chronic malnutrition due to other causes

Although the various liver function and related laboratory tests currently in general use are valuable in establishing the nature and degree of hepatic disease as well as in following its course, they must be regarded as supplements to, not substitutes for, careful clinical observation The development of punch biopsy technique has added greatly to our diagnostic armamentarium

JAUNDICE

This is discoloration *of tissues secondary to excessive concentration of bilirubin* in the blood As already noted, it is best seen in the skin and sclerae, can be

readily overlooked in artificial light, and may be confused with discoloration due to other causes (*see* Chap 4). Depending on the amount of bilirubin present, the hue may be pale or deep yellow, or orange, in long-standing obstruction it may be yellowish-green or green. Minimal hyperbilirubinemia not sufficient to create visible jaundice can be detected by blood chemical studies (*latent jaundice*) Generalized *itching is a common and distressing symptom*, especially in the obstructive form, sometimes in the parenchymal When either is severe, bradycardia or mental depression is likely, these are not encountered in the prehepatic form The various types of jaundice and their more common causes are as follows

Prehepatic (Retention). Increased hemolysis produces, in the blood, such an excessive amount of bilirubinglobin that it cannot normally be excreted by the liver Some observers believe that an associated hepatic cell variant, causing elevation of liver threshold for bilirubin excretion, may be a factor Since bilirubinglobin (*indirect reacting* bilirubin) is not excreted by the kidneys, the urine, in contrast to the other forms of jaundice described below, retains its normal color Normally excreted bile reaches the duodenum so there is no change in the color of its contents or of the stools, as may occur in parenchymal or posthepatic jaundice Prehepatic jaundice is seen in

1 Jaundice of the newborn This is a physiologic process occurring in 50 per cent or more of newborn infants and marked by jaundice of varying degrees starting on the second or third day of life, beginning to decline within a week or so, and disappearing after a fortnight

2 Hemolytic anemias, such as familial or acquired hemolytic anemia, sickle-cell anemia, Mediterranean anemia, and erythroblastosis fetalis (*see below*)

3 Pernicious anemia The characteristic lemon-yellow tint often seen in these cases is due to a combination of pallor and mild jaundice

4 Severe malaria

5 Severe lobar pneumonia, streptococcus septicemia, or other overwhelming infection In these cases, the jaundice may be of both prehepatic and intrahepatic origin, the latter resulting from toxic action of the infecting agent on the liver cells

6 Massive pulmonary infarction, sometimes

7 Incompatible transfusion

8 Toxic action of certain drugs, such as the sulfonamides and phenylhydrazin

9 Massive hematoma, usually the result of trauma

Parenchymal (Intrahepatic Regurgitation). Necrosis of liver cells and obstruction of small bile radicals by edema, inspissated bile and detritus cause regurgitation back into the blood stream of bilirubin excreted into the bile canaliculi by those cells which are still functioning This changed (*direct reacting*) bilirubin is deposited in the tissues to cause *the jaundice Retention* of bilirubinglobin due to impairment and diminution of liver cells may be a contributing factor. Since the kidneys are capable of excreting direct reacting bilirubin (in contrast to the indirect reacting bilirubin which they cannot excrete) *some of it will* appear in the urine and give it a bile color If intra-

hepatic edema and inflammation are sufficiently pronounced, so little bilirubin will reach the intestinal tract through the normal pathway that the stools become pale or clay-colored. Parenchymal jaundice is most commonly seen in:

1 Acute intrahepatic processes resulting from, (a) infection, as in infectious or homologous serum hepatitis, mononucleosis, cholangiolitic hepatitis, Weil's disease, yellow fever, and perhaps syphilis, (b) some hepatotoxic agent such as arsenic, chloroform, cinchophen, carbontetrachloride, phosphorus, or trinitrotoluene, or that elaborated in toxemia of pregnancy; (c) anoxemia, as in chronic passive congestion or marked anemia

2 Cirrhosis of liver Mild jaundice is sometimes a feature of well-established *portal* cirrhosis. The urine may show bilirubin but bile in stools and duodenal contents is rarely appreciably diminished In *biliary* cirrhosis jaundice is usually persistent but may wax and wane; color changes in urine and stools parallel the variations.

3. Diffuse neoplastic disease of the liver, such as lymphoma or carcinoma. Jaundice is a late manifestation occurring only when the bulk of liver parenchyma has been replaced by tumor Intrahepatic bile duct obstruction is also a factor

4. Suppurative cholangitis with ascending infection Since this disease, usually a complication of cholelithiasis or stricture, is marked by inflammation of the biliary ducts, the jaundice is primarily posthepatic However, it can be regarded as partly intrahepatic since bilirubin excretion is further hampered by impairment of liver cells due to associated stasis and infection In the late stages, bile may be absent from stools and duodenal contents Marked constitutional symptoms are present.

5 Suppurative pylephlebitis Mild jaundice is the rule It too probably represents a combination of parenchymal inflammation, and obstruction of intrahepatic ducts by inflammatory edema or pressure from abscesses within the liver substance

Posthepatic (Obstructive Regurgitation). Flow of bile into the duodenum is hampered by obstruction of the common or, less often, hepatic duct Direct reacting bilirubin is consequently resorbed from the biliary tree back into the blood stream and jaundice appears Since this changed bilirubin can pass through the kidneys the urine is bile-stained The stools are pallid or clay-colored due to interference with normal flow of bile into the duodenum In cases of short duration, impairment of liver function is not striking unless there is an associated cholangitis, usually indicated by chills, fever, and leukocytosis However, since back pressure in the biliary tree causes some impairment of liver cell function, diminished excretion of indirect reacting bilirubin contributes to the jaundice Later, greater damage to liver cells occurs and adds a greater element of intrahepatic jaundice. In long standing obstruction, the patient's color may be almost green, presumably because of change in the bile pigment from yellow or orange bilirubin to greenish biliverdin. Severe pruritus is most likely to occur in the posthepatic case and sometimes appears before jaundice is present. Obstructive jaundice is most likely due to·

1 Stone in common duct. Jaundice is likely to wax and wane The liver is not appreciably enlarged except in the long-standing case or when obstructive cirrhosis has developed Gallbladder is not enlarged Bouts of fever and/or pain resembling biliary colic are likely The urine is discolored and the stools clay-colored only during periods of pronounced obstruction

2 Tumor Cancer of head of pancreas, ampulla of Vater, or common or hepatic duct causes progressive, eventually intense, sometimes painless obstructive jaundice In cancer of the ampulla, waxing and waning is possible, intermittent necrosis with sloughing of a segment of the tumor may temporarily permit passage of bile, but regrowth will again create obstruction. In well-established block of the common duct, the gallbladder is distended and may be felt as a smooth, rounded tumor in the right upper quadrant, the liver is enlarged The urine shows marked discoloration, the stools are pallid or clay colored, and bile in the duodenal contents is minimal or absent. Loss of flesh and strength is rapid Jaundice is a later manifestation in cancer of the gallbladder; the organ may be felt because of tumor infiltration but is not necessarily distended In metastatic or primary cancer, or lymphoma, jaundice may be created by lymphnode impingement on the extrahepatic duct, neoplastic obstruction of intrahepatic ducts, or neoplastic replacement of the bulk of liver parenchyma The liver is large, whether the gallbladder becomes distended depends on the site of the block

The substance of *Courvoisier's law* is as follows When complete obstruction of the common duct is due to tumor, the gallbladder is distended and palpable; when due to stone it is not, because gallstones are so often associated with a thickened, contracted gallbladder There are so many exceptions to this rule that it is no longer regarded as of much diagnostic significance

3. Enlarged lymphnodes Lymphoma or metastatic tumor involving the periportal nodes may create obstructive jaundice by pressure on or invasion of the common or hepatic duct.

4. Inflammation Edema or cellular infiltration secondary to an inflammatory process in a nearby structure, especially the gallbladder or pancreas, may create temporary, partial, or complete obstruction of a major duct Occasionally block is caused by a chronic low-grade inflammatory process in the duct itself (*sclerosing cholangitis*), this is most likely to be encountered with a history of previous gallstones, whereupon it is regarded as due to inflammation initiated by previous passage of a stone.

5. Traumatic stricture Usually the result of a surgical misfortune, this may not be evident until weeks or months after the operation.

6 Congenital atresia or narrowing of common or hepatic duct This is encountered in early life and is fatal unless some palliative surgical procedure can be effected

7. Chemical cholestasis. In recent years a picture which clinically and by laboratory studies closely resembles obstructive jaundice has been seen following therapeutic administration of chlorpromazine, methyl testosterone, thiouracil, and certain other drugs. No obstructive lesion of a major duct is

present. Microscopic examination of liver tissue shows bile stasis of the cholangioles but no significant hepatocellular changes.

Although laboratory studies are without the scope of this book, it has seemed worthwhile to include a summary of the more important findings which are helpful in differentiating the various forms of jaundice. The reader must remain alert to the fact that none of these is necessarily definitive; overlapping is often encountered as, for example, when parenchymal liver disease develops secondarily to long-standing posthepatic obstruction, or when intrahepatic obstruction due to edema and inflammation becomes a complicating factor in acute hepatitis. (*See* Fig. 31.2.)

	URINE BILE	URINE UROBILINOGEN	DIRECT SERUM BILIRUBIN	INDIRECT SERUM BILIRUBIN	SERUM FLOCCULATION TESTS*	SERUM ALKALINE PHOSPHATASE
Prehepatic	Absent	Normal to slightly increased	Normal	Increased	Normal	Normal
Parenchymal	Present	Increased (during acute phase of hepatitis may be *decreased to* absent)	Increased	Increased	Positive	*Moderately increased*
Posthepatic	Present	Decreased to absent (in cholangitis may be increased)	Increased	Increased	Normal (in prolonged obstruction may become positive)	Greatly increased

	SERUM ALBUMIN	SERUM GLOBULIN	SERUM CHOLESTEROL	SERUM CHOLESTEROL ESTERS *% of total*	SERUM CHOLINESTERASE	SERUM BROMSULFALEIN RETENTION†
Prehepatic	Normal	Normal	Normal	Normal	Normal	Normal (may be increased in moderate to *severe anemia*)
Parenchymal	Normal to decreased	Normal to increased	Normal to decreased	Decreased	Decreased	Increased
Posthepatic	Normal	Normal	Increased	Normal	Normal	Increased

* Numerous flocculation tests have been developed. At the Massachusetts General Hospital the cephalin-cholesterol flocculation and the thymol turbidity and flocculation methods are preferred.

† Although the bromsulfalein test is the most sensitive index of liver function, it has little value in differentiating the various causes of jaundice.

FIG. 31.2. Chart of important laboratory findings in various types of jaundice. (Prepared with assistance of Dr. Perry J. Culver.)

Enlargement of Liver

Palpation is used to locate the lower border, provided it extends below its normal level. Percussion locates the upper border and may, by disclosing obliteration of Traube's semilunar area of tympany, point toward possible enlargement of the left lobe. Except for its use in determining whether a liver is shrinking (*see above*), percussion of the lower border is unreliable since the note at this level is masked by that of adjacent intestine. Estimation of liver size by x-ray is undependable and at times misleading, because the three dimensions are of equal importance, but only two can be reliably reproduced on conventional films For example, a thin tongue of liver parenchyma extending downward (*Riedel's lobe*) might create a shadow suggesting enlargement in spite of the fact that this variant represents no significant increase of liver substance

Hepatic enlargement can be assumed if the lower border is readily felt below the costal margin, provided the three following sources of confusion can be excluded

1 Downward displacement Percussion will show the upper border of flatness to be below its normal level—the sixth intercostal space in mid-clavicular line At times percussion here is not helpful because the liver has been displaced by an intrathoracic disturbance such as pleural effusion or pneumothorax which obscures the note reflected from the liver In pleural effusion, for example, the flat note due to fluid merges with that of the liver; one cannot detect a line of demarcation



Fig 31 3 Unimanual palpation of liver Correct position of examiner's fingers, hand and forearm



FIG 31 4 Bimanual palpation of liver Correct position of examiner's fingers, hands and forearms

2. Elongated right lobe In the narrow-framed person, the liver may be elongated so that its right lobe normally extends below the costal margin

3. Riedel's lobe (*see* Chap. 29)

Palpation of Liver. The fundamental rules for abdominal palpation apply (*see* Chap 29) *Slight enlargement or displacement should be detectable by* one of the following methods, both of which must be tried in the doubtful case·

1. Sitting or standing at the patient's right shoulder, the examiner rests his palm in a relaxed position on the lower part of the thorax and hooks his fingers without too much pressure upward, toward, or actually under the right costal margin The patient is instructed to inspire deeply through his mouth. If the organ is slightly enlarged or displaced downward, the edge will be felt as it descends with inspiration.

2 The examiner sits or stands opposite the upper right abdomen, slips his left hand under the patient's loin at the lower border of the thorax and places his right hand on the abdominal surface, with the fingers just below and parallel to costal margin. The patient is again instructed to take a deep breath. As he does so, the left hand is pushed upward toward the right hand while the latter, with light pressure maintained, is permitted to rise with the upward movement of the abdominal muscles, the edge should be felt as it descends.

In both methods, one must feel, not at a single point, but all along the border of the thoracic cage and in the epigastrium before regarding his examination as negative

If the edge is not felt in this zone, the same two procedures must be tried

with the palpating hand at successively lower levels of the abdomen. Otherwise moderate or great enlargement is likely to be overlooked because firm direct pressure on the anterior surface of the liver will force the organ posteriorly so that it escapes detection.

In the presence of ascites ordinary palpation is ineffectual, but it is often possible to feel an enlarged liver by ballottement (*see* Chap. 29). When the fluid is removed, the flaccidity of the abdominal wall permits the organ to be easily felt by the usual methods. Ballottement may also be helpful in an obese or distended abdomen

One should note whether the edge is sharp or rounded, the surface smooth, uneven or nodular, and the consistency normal or unusually hard or soft. If the edge is smooth, extends across the right side of the abdomen, and descends with inspiration, it is rarely mistaken for any thing else, if it is irregular, nodular, and not easily traced across the abdomen, it is often difficult to determine whether one is feeling liver, a tumor of stomach or other organ, or a mass of matted omentum and bowel

Causes of Hepatic Enlargement. In the approximate order of their frequency, the more common causes are

1 Fatty infiltratiaon Slight to moderate enlargement is the rule In obesity or alcoholism, storage of excess fat is responsible, in the former, the liver may not be palpable because of the fat abdominal wall In wasting diseases, especially cancer, tuberculosis, and ulcerative colitis, fat in other parts of the body, before being utilized, is temporarily transferred to the liver, thus increasing its size The edge is smooth, rounded, and soft

2 Passive congestion Usually due to congestive heart failure, rarely, to constrictive pericarditis or impairment of return blood flow from other cause such as obstruction of hepatic vein, enlargement is proportionate to the severity and duration of the underlying ailment The border may extend 1–10 cm below costal margin or even lower, and is sharp or rounded, tenderness is variable depending on the acuteness of the process In a long-standing case, because of increased fibrous tissue, the surface is hard, non-tender, and perhaps somewhat irregular In clinical cardiac cirrhosis (*see below*) the liver may be small

3. Hepatitis Enlargement is usually slight to moderate, the edge, extending 1–6 cm below the costal margin, is smooth and somewhat tender In the most severe cases, eventual shrinkage due to diffuse necrosis is likely In the chronic form the size may vary from time to time, depending on such factors as physical exertion, *intercurrent infection, and alcoholic intake*

4 Cirrhosis The liver is moderately enlarged except in the late stages of post-necrotic or cardiac cirrhosis, when it is likely to become small The edge feels firm, smooth, and is non-tender With the exception of *syphilitic cirrhosis, irregularity of* surface is not, contrary to some teaching, sufficiently pronounced to be detectable by palpation

5 Malignant disease. Metastatic carcinoma or sarcoma, less often, primary carcinoma, is probably the most common cause of a huge liver. The organ may

enlarge rapidly and presents a hard, irregular, sometimes definitely nodular surface. If nodules can be felt, the diagnosis proves almost invariably to be malignant disease. In lymphoma, moderate enlargement with rounded and firm edge is the rule but nodules can occasionally be felt.

6 Biliary tract obstruction Liver size depends on the duration and degree of obstruction. When the latter is complete and long-standing, the edge may extend well below the umbilical level In the long-standing case, secondary infection and cirrhosis are often contributing factors.

7. Leukemia. In acute or chronic lymphatic leukemia, liver size is normal or slightly increased Chronic myelogenous leukemia is one of the causes of extreme enlargement; the edge is smooth, firm, and often extends below the umbilicus

8 Syphilis In the eruptive stage of early syphilis, the liver is occasionally slightly enlarged. In late disease, it may be uniformly enlarged and firm, due to presence of numbers of small gummas and diffuse interstitial fibrosis Large gummatous areas may be felt as tumor like masses With healing, the organ becomes distorted by deep, depressed scars which give an irregular or lobulated surface (*hepar lobatum*) mistakable for that of nodular malignant disease. In congenital syphilis, slight uniform enlargement may be found, healing is accompanied by the same changes as in acquired syphilis but lobulation may not be striking

9. Rickets In rickets and other nutritional disturbances in children, the liver may be easily felt, consistency is normal Although some degree of enlargement is usually present, poor tone of abdominal musculature and outward flare of lower thoracic cage undoubtedly contribute to the easy palpability

10 Amebiasis Amebic *hepatitis* is characterized by diffuse, slight to moderate liver enlargement, with tenderness Amebic *abscess*, usually solitary, sometimes multiple, is likely to extend toward the superior surface on the right and push the diaphragm upward, whereupon expansion can be detected only by x-ray, occasionally the lower border will be pushed downward far enough to be palpable Rarely, an abscess points on the lower surface and can be felt as a smooth, rounded, tender mass

11 Acute suppurative liver disease Multiple abscesses, secondary to pyogenic infection elsewhere, cause moderate enlargement and tenderness

12 Acute suppurative cholangitis The liver is tender and usually somewhat increased in size

13 Amyloidosis Uniform, slight to moderate enlargement and a smooth surface without pain or tenderness are the rule Signs of hepatic insufficiency may eventually develop A chronic suppurative or inflammatory process elsewhere, such as osteomyelitis, tuberculosis, or rheumatoid arthritis is usually found, rarely, the disease is idiopathic or occurs in association with multiple myeloma Amyloid deposits are not confined to the liver but appear also in the spleen, kidneys, lungs, heart, skeletal muscles, and other structures, and may, if sufficiently concentrated in a vital organ, create parenchymal insufficiency.

14. Hydatid cyst Echinococcus cyst on the anterior surface may form a



Fig 31 5 Swelling of abdomen due to enlargement of liver and spleen, and ascites in amyloidosis secondary to chronic suppuration in draining sinuses from tuberculosis of spine

rounded, tense, somewhat elastic, non-tender mass palpable in the right upper quadrant When one develops on the posterior surface, expansion is upward and signs are similar to those of subphrenic abscess or pleural effusion at the right base *Hydatid thrill*, though absent in many cases is, when encountered, a most distinctive and clear-cut sign If the examiner presses quickly downward on the suspected area and then holds his hand flat against it, he will feel a peculiar vibratory sensation as the daughter cysts, displaced by the inital shove, rebound upward in the major sac Eosinophilia is often a helpful sign If, as sometimes happens, one or more cysts become calcified, circinate shadows will be observed by x-ray.

15 Schistosomiasis. Because of the frequency of schistosomiasis in troops returning from endemic areas, and in immigrants from Puerto Rico, enlargement of the liver in *Schistosoma japonicum* and *Schistosoma mansoni* must be mentioned Proliferation and fibrosis secondary to deposition of eggs result in progressive enlargement of liver and some enlargement of spleen Eventually the picture becomes that of portal cirrhosis with diminution in liver size, congestive splenomegaly, and ascites

16 Kala-azar Also deserving mention because of its appearance in military personnel returning from endemic regions, this disease shows, in its later stages, enlargement of the liver from reticulo-endothelial proliferation (*see below*)

Hepatitis

This term refers to diseases marked by diffuse inflammation of liver parenchyma brought about by a systemic infectious process, absorption of some toxic agent, or a nutritional disturbance When mild, the changes are probably limited to edema and fatty degeneration with slight systemic response and minimal or absent clinical jaundice The more severe process is marked by some degree of liver cell necrosis and functional impairment, more pronounced jaundice and greater systemic disturbance.

Acute Infectious Hepatitis. Known also as *epidemic hepatitis* and formerly

but no longer as *catarrhal jaundice*, this disease can occur sporadically but is more likely to appear in mild or major epidemics, especially where persons are concentrated, as in military installations and schools. A filtrable virus is well-established as the etiologic agent Most authorities agree that transmission is usually by ingestion of fecal-contaminated food or water In contrast, hemologous serum hepatitis (*see below*) is acquired parenterally Although the subject of etiology is still confused, currently it is known that there are at least two strains of virus, one of which is responsible for most of the acute infectious cases and the other for most of the homologous serum cases. Whether the virus of the former can be transmitted parenterally or that of the latter non parenterally remains unsettled Although extremely important from the epidemiologic standpoint, these etiologic considerations are of little clinical consequence The term *viral hepatitis* is used to include these two forms of hepatitis and sometimes also that associated with mononucleosis

The incubation period of the acute infectious form varies from 2–6 weeks. Onset is marked by fever, anorexia, headache, malaise, lassitude, nausea and perhaps vomiting, and mild diarrhea, all of a few days' duration Following this prodromal period jaundice appears, accompanied by accentuation of symptoms, malaise, anorexia, nausea, and vomiting predominate. Attacks of acute abdominal pain may occur The liver becomes slightly enlarged, and tender to direct or compression palpation, increasingly so if the patient is permitted to be active The spleen is palpable in about one-third of cases; generalized lymphnode enlargement, especially of the lower cervical chains, is not unusual The urine becomes bile-colored, the stools pallid or clay-colored Following slight leukocytosis in the predromal phase, the blood shows leukopenia chiefly of polymorphenuclear cells In the average case, symptoms continue and jaundice deepens for a few days to two weeks or longer after which the former disappear quite suddenly and the latter begins to fade. Diuresis and reappearance of appetite are often the first clues to beginning improvement Enlargement of liver, spleen, and lymphnodes gradually recedes Convalescence is slow and, unless the patient is carefully guarded, especially from too rapid resumption of activity and from use of alcohol, relapse is a distinct possibility Although this picture is the usual one, many cases pursue a much longer course with jaundice and other manifestations persisting for weeks; they may develop into chronic hepatitis Some patients who appear to have recovered completely end up with cirrhosis years later.

Mild cases with minimal symptoms and absent or minimal jaundice are probably more common than is generally supposed. Here, recovery is the rule but it is quite possible that some of them, because of inadequate treatment due to non-recognition of the disease, subsequently develop chronic hepatitis or cirrhosis

The most severe cases, instead of showing improvement after a week or so, pursue a steadily downward course with gradual but sometimes abrupt appearance of profound liver failure Jaundice becomes intense; weakness, prostration, headache, restlessness, nausea, and vomiting are severe Hepatic pain,

sometimes colicky, and usually liver tenderness are evident. The characteristic "mousy breath" (*fetor hepaticus*) may be observed Most striking is the development of central nervous system changes: somnolence, disorientation, euphoria or other personality deviation, tremulousness, and eventually delirium, convulsions, and coma The liver, previously enlarged, becomes smaller; this change may be demonstrable by day-to-day palpation and percussion Mucous membrane and intracutaneous bleeding, edema, ascites, anemia, and oliguria are other late or terminal features Many cases are fatal. Some, however, regenerate sufficient liver parenchyma to permit temporary recovery but cirrhosis may develop subsequently. In others, recovery is partial, the patient remains in a state of subacute or chronic hepatitis with persistence of jaundice or other indications of trouble.

Occasionally one encounters an acute fulminating form of infectious hepatitis ushered in by high fever, generalized aches, severe vomiting, epigastric discomfort, early appearance of cerebrospinal changes and bleeding diathesis, and sometimes pronounced leukocytosis Jaundice may be severe, mild or absent, its degree depending on the duration of the illness Ascites and edema occur in all but the shortest cases Death within 1 days to 2–3 weeks is the rule

Homologous Serum Hepatitis. Here the hepatitis is acquired by the parenteral route. The virus may be introduced into the blood stream or tissues by blood or a blood product, or transmitted from a known or unknown carrier by an inadequately sterilized needle, syringe or other piece of equipment used in a diagnostic or therapeutic procedure

Homologous serum hepatitis has an incubation interval of 2–5 months as opposed to the shorter period of the acute infectious type Onset tends to be insidious and afebrile Mild symptoms, such as malaise, anorexia, tendency to diarrhea, right upper quadrant or lumbar ache may occur from time to time during the incubation period. Urticaria and arthralgia are sometimes encountered. Once the disease becomes established, the picture is similar to that described above but the course may be more serious and protracted, perhaps, in some instances, because the procedure which introduced the infection was performed in a patient already suffering from some other illness

Mononucleosis. Jaundice and usually some enlargement of the liver indicative of mild to moderate hepatitis are sometimes encountered in infectious mononucleosis. Even in the absence of these signs, abnormal liver function tests reflecting some degree of parenchymal impairment are almost always found.

Chronic Hepatitis. A few patients with acute infectious or hemologous serum hepatitis, and, rarely, one with hepatitis complicating mononucleosis, fail to recover completely within the usual period but remain beset by continuous or intermittent manifestations of low-grade trouble They are often incorrectly diagnosed as psychoneurotics. Exacerbations and remissions may be related to such factors as exertion, intercurrent infection, and use of alcohol. The usual complaints are lassitude, fatigability, digestive disturbances,

especially fat intolerance, and right upper quadrant or lumbar aching. Jaundice is often absent. The liver may be somewhat enlarged and tender. These variants are increased by activity: a liver which is non-palpable when the patient has been inactive may be easily felt at the end of a busy day. As a rule, *hepatic function studies show impairment but biopsy may be necessary* for diagnosis. The ultimate outcome is complete recovery, cirrhosis, or death from liver failure during an acute exacerbation.

Occasionally in prolonged disease, cholangiolitic rather than hepatocellular impairment appears to predominate. These cases show such evidence of post-hepatic jaundice as itching, hypercholesteremia, and hyperphosphatasemia as to make *them readily confusable with extrahepatic obstruction. In fact,* to distinguish between the two may be impossible without cholangiography through a small incision, or surgical exploration. They usually proceed to cirrhosis.[1]

Toxic Hepatitis. The cause is absorption of some chemical agent, of which the most important are chloroform, carbon tetrachloride, arsenic, cincophen, dinitrophenol, trichlorethylene, phosphorus, and trinitrotoluene. Individual sensitivity appears to be a factor in determining the extent of liver damage; an amount of the chemical which in one person will cause no reaction may, in another, produce severe parenchymal disease. Alcohol alone probably does not cause any acute liver changes but will seriously aggravate an existing hepatitis. Acute toxic hepatitis is occasionally seen in association with pernicious vomiting of pregnancy. At times one cannot decide whether a case is toxic or viral.

Onset is afebrile and fairly sudden but otherwise similar to that of the infectious type. Nausea and vomiting are prominent. Jaundice appears early; the liver is tender and somewhat enlarged. The end result may be complete recovery, severe necrosis with either partial recovery or death, or eventual development of cirrhosis.

Syphilis. In early syphilis, jaundice may reflect a mild hepatitis due to the infection. However, most observers now believe that hepatitis occurring in early syphilis is due, not to the disease itself or to the toxic effect of arsenic (as was postulated when arsenicals were widely used therapeutically), but more likely to parenteral transmission of virus by contaminated needle or syringe. Jaundice attributable to late syphilis is rare.

Weil's Disease. This acute infection, caused by *Leptospira icterohemorrhagiae, is encountered most commonly in* persons who come in contact with excreta of rats, such as workers in abbatoirs, tunnels, sewers, and fish piers. Onset is abrupt, with chills, fever, prostration, headache, nausea, vomiting, severe myalgia, especially of the calves, and sometimes signs of meningeal irritation. Conjunctival injection is the rule. About the fifth or sixth day, temperature falls abruptly, and jaundice, with perhaps enlargement and tenderness of the liver, appears, sometimes bleeding tendency and indications of renal irritation develop. The spleen is not palpable. These later manifestations

are sometimes so minimal that the nature of the illness is unrecognized. Recovery is the rule but minor relapses are likely. Weil's disease is distinguished from acute infectious hepatitis by the severe systemic reaction at onset, and presence of conjunctivitis, muscle cramps, and leukocytosis Diagnosis is established in the early stage by recovering the organism on guinea pig inoculation of the blood Sometimes it can be made on dark-field examination of centrifuged blood After the first few days, the organism disappears from the blood but may, by similar methods, be found in the urine After approximately the tenth day serum agglutination tests are positive.

Yellow Fever. Fortunately rare in this country, yellow fever is an acute infectious disease producing severe changes in the liver, renal tubules, and blood capillaries Milder cases show swelling and fatty degeneration of liver parenchyma, the more severe ones, extensive necrosis Onset is sudden with fever, malaise, muscular pains, and usually nausea and vomiting The outstanding signs which appear within 1-3 days are hemorrhage, especially from the gingivae, nose and gastro-intestinal tract, jaundice with perhaps slight liver enlargement, and evidence of acute nephritis As a rule, death occurs or recovery begins within the first week Although fever and other systemic signs end fairly abruptly, jaundice and albuminuria may persist for several weeks but permanent renal or hepatic damage is not common

Acute Suppurative Liver Disease

The diagnosis of liver abscess depends more on the history and general clinical picture than on local signs. It must be suspected when septic fever and leukocytosis appear with absent or minimal localizing symptoms or signs in the course of some suppurative process in the abdomen, especially appendicitis, after long-continued biliary obstruction, or when the possibility of amebic infection exists

Multiple Abscess. The usual causes are.

1 Blood stream infection In general septicemia or pyemia, features pointing toward hepatic involvement are not striking The picture is that of severe general sepsis with intermittent fever, chills, sweats, prostration, and leukocytosis Liver involvement is to be suspected if the patient complains of pain over the liver or in the right lumbar region, or one finds direct or compressive *liver tenderness* or lumbar tenderness. Pain in the shoulder or local pain increased by inspiration may be created by involvement of the diaphragmatic surface of the liver Jaundice, usually mild, is a late manifestation

2 Pylephlebitis Secondary suppuration in the liver by way of the portal vein is suggested when the picture just described develops in a case of acute infection of an intra-abdominal structure whose blood supply drains into the portal system It is most likely to be encountered following suppurative appendicitis Rarely, a single abscess will form.

3. Acute suppurative cholangitis Extension upward into the liver with formation of small abscesses is almost inevitable. No additional symptoms appear, the picture remaining that of the underlying cholangitis.

4. Amebiasis. Although amebic abscess of the liver is usually single, occasionally more than one will develop

Solitary Abscess. Almost always secondary to intestinal amebiasis, this may appear during an acute attack of dysentery, or at any time—even years—thereafter. The intestinal infection may have been so mild as to have gone unrecognized; often in chronic amebic abscess no history suggesting previous amebiasis can be elicited Amebic abscess has a predilection for the upper part of the right lobe and tends to extend upward, eventually reaching the diaphragmatic surface and perhaps giving a picture confusable with a basal pleuropulmonary process When it develops acutely, it is usually preceded by amebic hepatitis

Amebic Hepatitis This is characterized by septic fever with chills and sweats, weakness, loss of weight, leukocytosis, and hepatic pain, tenderness, and usually some enlargement. The pain may be dull or sharp, is felt over the liver, in the epigastrium or right costovertebral region, and may be referred to the lower thorax and shoulder Aggravating factors are cough, change of position, and, especially, jarring Tenderness may be brought out only by the compression palpation or fist percussion Nausea and vomiting occur in about a third of the cases, jaundice is relatively uncommon and rarely striking. Dulness and diminished respiratory sounds at the right base due to elevation and splinting of the diaphragm are likely

Acute Amebic Abscess This presents a similar picture In fact, many authorities believe that, as a rule, a diagnosis of acute abscess can be made only when anti-amebic therapy in a case of amebic hepatitis has failed to create improvement within a few days Suggestive signs of abscess formation are increasing systemic response, cough, pain in the right shoulder, an area of localized tenderness over the liver and sometimes, a visible or palpable bulge over or near the liver Associated prostration, acute pain, and upper abdominal muscular spasm hint at some other acute intra-abdominal insult such as ruptured ulcer or acute pancreatitis X-ray is helpful in showing diaphragmatic elevation, irregularity, or doming, associated pleuropulmonary changes at the right base, and in excluding subphrenic collection of air.

Chronic Amebic Abscess Although sometimes occurring after amebic hepatitis, chronic amebic abscess is more likely to appear insidiously—months or years after an attack of dysentery or without history of previous intestinal trouble The patient may complain of malaise with anorexia and perhaps loss of weight for a considerable time before localized manifestations appear Because of the tendency of the amebic lesion to extend toward the superior surface of the liver, pain in the shoulder or thorax with perhaps evidence of some changes at the right base may lead to a mistaken diagnosis of pulmonary tuberculosis Fever and leukocytosis are variable. As a rule, irregular fever will develop so that amebic abscess must always be considered in any obscure case of fever Signs pointing to the liver as a possible site of trouble are deep pain, especially on jarring, tenderness on compression or fist percussion, and sometimes fingerpoint tenderness in an overlying intercostal space. Rarely, extension downward and anteriorly will occur, enlargement of the liver with

perhaps swelling and fluctuation below the costal margin will then be found. Upward displacement of diaphragm will cause signs resembling right pleural effusion, subphrenic abscess, or consolidation of the right base. Diaphragmatic motion, as determined by Litten's shadow and x-ray is less restricted in abscess than in the others. As in acute abscess, x-ray will show elevation, irregularity, or doming of the diaphragm, and perhaps indications of a pleuropulmonary complication.

Acute rupture through the diaphragm will cause empyema but if extension proceeds slowly so that pleural adhesions form between lung and diaphragm, the plural cavity may be by-passed and a lung abscess created.

Cirrhosis of Liver

Various classifications of this disease have been proposed We regard the following as satisfactory for ordinary purposes In most cases, it is possible to identify the type of cirrhosis but there are times when, because of the occasional tendency for one form to shade into another, one cannot be certain of the exact nature of the process

1 Alcoholic } Portal
2 Postnecrotic }
3 Obstructive (extrahepatic) } Biliary
4 Cholangiolitic (intrahepatic) }
5 Syphilitic (hepar lobatum)
6 Pigment (hemochromatosis)
7 Cardiac
8 Schistosomiasitic

Alcoholic Cirrhosis. This is the type originally described by Laennec In most cases a history of prolonged and excessive indulgence in alcohol is obtained, but by no means do all generous imbibers develop the disease Most observers believe that alcohol *per se* is not the sole or direct cause of cirrhosis but that some other factor related to alcoholism such as failure to eat or properly to metabolize ingested food is at fault All agree, however, that any form of parenchymal disease of the liver will be made worse by alcohol

Usually encountered in middle life or later, alcoholic cirrhosis due to the great reserve of the liver parenchyma, may not manifest itself until it is far advanced Often its presence is unknown until indications of failure occur, perhaps precipitated by a drinking spree, an intercurrent infection, or some form of stress In other cases the first indication is a sign of interference with local circulation or protein metabolism. hematamesis, melena, ascites, edema of the legs, or discovery of esophageal varices by x-ray

Prior to appearance of any of the above, the patient may have been bothered by symptoms such as fatigability, anorexia, dyspepsia, flatulence, a feeling of heaviness in the liver region, and sometimes nausea and vomiting, especially in the morning. The gastric symptoms, however, may be related more to an associated gastritis than to the cirrhosis itself In the earlier stages, physical examination will show a large, perhaps but not necessarily, tender liver; its

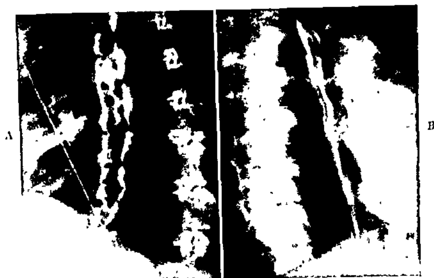


FIG 31 6 A Esophageal varices in a case of cirrhosis of liver Tortuous filling defects and widening of lumen due to inward projection of dilated veins
B For comparison with A, barium outlining folds of normal esophagus

edge is usually smooth and somewhat rounded, in contrast to the later phase when it is sharp and firm The enlargement is due to excessive fat. The spleen is palpable in many cases. Slight icterus or low-grade fever may be observed. As the process advances, the indications of parenchymal and local circulatory impairment become pronounced. The symptoms described above are more distressing, bouts of colicky pain suggesting gallstones may occur. The appearance of the patient becomes quite characteristic the face, thorax, and upper extremities are thin, the eyes sunken, watery, and slightly icteric, and the abdomen prominent because of ascites Jaundice of some degree may be evident. The liver is smaller than beforehand but usually palpable or, in the presence of ascites, ballottable. Although at this stage, its surface is undoubtedly granular or slightly nodular (*hobnail liver*), this variant, contrary to some teaching, cannot be detected through the abdominal wall Occasionally shrinkage is sufficient to make the organ non-palpable. The spleen is somewhat enlarged but difficult to feel in the presence of ascites; splenic friction may be heard or felt The veins of the abdomen are prominent but direction of blood flow is normal The legs are thin or edematous Enlarged or bleeding internal hemorrhoidal veins may be seen through the anoscope. Spider angiomatas and liver palms (*see above*) are common. Grayish-black pigmentation on the dorsums of the hands, forearms, and exposed surfaces of the neck may appear. In the long standing case, inability of the liver to conjugate estrogens causes, in the male, testicular atrophy, diminution of axillary hair, tendency toward female texture of skin and distribution of pubic hair, and perhaps gynecomastia; in the female, abnormal uterine bleeding may develop. Esophageal varices are often demonstrable by careful barium x ray studies, or

esophagoscopy Death can occur from total liver failure, hemorrhage from ruptured esophageal or hemorrhoidal vein, or from *E. coli* septicemia, a hazard to which the cirrhotic patient is susceptible Superimposed primary carcinoma is a not uncommon complication.

Postnecrotic Cirrhosis. Formerly known as *toxic cirrhosis*, this may be the end result of toxic or perhaps infectious or homologous serum hepatitis Some cases appearing without a history of former liver trouble may reflect unrecognized hapatitis occurring years previously In general, the clinical picture is similar to that of alcoholic cirrhosis In the earlier stages, the liver may be large and its surface irregular as a result of spotty distribution of islands of regenerated parenchyma; extensive regeneration in the left lobe may make it easily palpable. Later, the liver is much smaller and cannot be felt From the clinical standpoint the differentiation between alcoholic and toxic cirrhosis depends on the history. The former is favored if, in the late stages, the organ is still palpable

Obstructive (Extrahepatic) Cirrhosis This results from prolonged partial occlusion of the common bile duct by stone, postoperative traumatic stricture, or sclerosing cholangitis Due to impaired drainage, low grade infection with resultant damage to smaller ducts and parenchymal cells may be a factor There is usually a history suggesting previous gallbladder trouble and perhaps bouts of intermittent jaundice, chills, fever and pain Even in the absence of calculi, recurrent attacks of acute pain simulating biliary colic are not uncommon Jaundice is persistent but may wax and wane When it is intense and longstanding, the patient's color may be greenish-yellow to almost green It is in this form of cirrhosis that itching is most likely and most distressing, this symptom may precede visible icterus and other physical signs of trouble by as much as a year The liver is moderately to greatly enlarged, has a smooth edge and may be somewhat tender. The spleen is easily felt Ascites is not a feature except rarely in the terminal stage Xanthomata may appear on the skin (*see* Chap 3)

Once established, the disease is chronic and persistent even though the cause, such as stone in the common duct, has been removed The course is marked by periodic exacerbations of fever, pain, increased jaundice, and greater size and tenderness of the liver Death occurs from hepatic failure or intercurrent infection Occasionally periportal scarring may proceed to such a point that in the late stage the disease cannot be distinguished from portal cirrhosis

Cholangiolitic (Intrahepatic) Cirrhosis. Here the process, primarily involving the small bile ducts and surrounding portal areas, conforms to the picture originally described by Hanot Some observers regard it as an idiopathic disease Others believe that evidence is accumulating to support the premise that many, if not all, cases in this group result from previous hepatitis, a history suggesting the latter may or may not be obtained

The patient is usually beset by repeated attacks of malaise, digestive disturbances, jaundice and perhaps fever and leukocytosis which tend to increase in duration and severity as time goes on. Itching is prominent. The symptoms,

physical findings, and course are so similar to those of extrahepatic biliary obstruction that the two can be *differentiated only by cholangiography or surgical exploration.*

Syphilitic Cirrhosis. Late syphilis of the liver is usually manifested by formation *of one or more large gummas* which first appear in the form of tumor-like masses causing pain and tenderness; rarely, they are large enough to cause bulging in the epigastrium or right hypochondrium. In time, due to *scarring and fibrous tissue contraction,* the liver becomes *deeply lobulated,* its shape deformed and its edge irregular and distorted (*hepar lobatum*). As a rule, no symptoms referable to the liver are present. The diagnosis is made on the basis of the irregular edge or a mass, and positive laboratory studies.

Pigment Cirrhosis. Hemochromatosis, rare in females, is characterized by deposits of hemosiderin and hemofuscin in the liver and other organs, especially the skin, pancreas, and heart It is thought to be due to increased iron absorption secondary to an intestinal tract variant, the nature of which is not known It can also occur following multiple transfusions given for some chronic ailment over a long period, and very rarely from prolonged therapeutic ingestion of iron

The predominant clinical features may be related to any of the organs involved Bronze-like pigmentation of the skin, symptoms referable to the liver, or discovery of insulin-resistant diabetes may first call attention to the disease When present, the pigmentation usually involves all of the skin but is more pronounced on the extensor surfaces, especially of the forearms and hands It may be concealed by jaundice or confused with the pigmentation encountered in hepatic cirrhosis, sometimes it is absent The manifestations of liver involvement are similar to those of portal cirrhosis. Unless the altered color of the skin is quite evident, this diagnosis is apt to be made and the underlying cause overlooked. Testicular atrophy and loss of secondary sex characteristics are likely to occur early. Their detection under any of the circumstances just described *should encourage one to entertain the diagnosis of hemochromatosis.* Death from liver failure or terminal infection is the rule. In a few patients indications of myocardial involvement—abnormal rhythm, dilatation and hypertrophy, *and progressive failure—predominate Such cases are often wrongly* diagnosed idiopathic cardiac failure (*see* Chap. 22) The diagnosis of hemochromatosis can usually be confirmed by biopsy of skin or liver, or demonstration of elevated serum iron and iron-binding capacity.

Cardiac Cirrhosis. From the pathologic standpoint, most cases of recurrent congestive failure may be said to show some degree of hepatic cirrhosis resulting from increased fibrosis secondary to chronic passive congestion. The term *cardiac cirrhosis* should be applied only to those cases of heart disease in which fibrosis and destruction of liver parenchyma have proceeded to such a point that a picture resembling late portal cirrhosis is found. This variant is most likely to occur in patients who have had repeated episodes of congestive failure, especially when they are due to hypertension, or to rheumatic disease, particularly if tricuspid involvement is present. An early stage may be postulated

when one finds, in association with recurrent congestive failure, a large, nontender liver, elevated venous pressure, and perhaps ascites. Later, the striking features are a small, non-palpable liver, splenomegaly, ascites, and pronounced elevation of venous pressure. *Post mortem* one finds portal cirrhosis, central cirrhosis, a combination of the two, or diffuse, patchy fibrosis.

Schistosomiasitic Cirrhosis. In the late stages of *S. japonicum* or *S. mansoni* infestation, replacecent of liver parenchyma by fibrous tissue creates a clinical picture similar to portal cirrhosis, including small liver, large spleen, and ascites

Malignant Disease of Liver

Primary Carcinoma. Although sometimes appearing independently, this is most apt to be superimposed on cirrhosis It may appear as a large, single lesion, multiple smaller nodules, or diffuse infiltration of liver substance Primary cancer must be suspected, especially in a case of cirrhosis, if the liver begins to enlarge, nodules become palpable or a mass appears, particularly if there is no evidence of primary tumor elsewhere Also to be viewed with suspicion is the patient with cirrhosis who shows rapidly developing cachexia, weakness, and weight loss, in the absence of striking clinical and laboratory indications of parenchymal impairment X-ray may show a characteristic picture consisting of evidence of cirrhosis—small or large liver, large spleen and esophageal varices—and a mass in the liver region which may displace intestine or, more likely, create a local bulge of the diaphragm. Evidence of metastatic disease in the lungs, or an abdominal mass secondary to lymphnode or mesenteric involvement may be found.

Metastatic Carcinoma. This is common The most likely sites of the primary lesion are stomach, colon, pancreas, breast, uterus, and ovary. Although early symptoms may reflect the primary disease often those related to the liver appear first. The patient will complain of indigestion, anorexia, nausea, vomiting, and perhaps local pain The last-named, if not present at the start, eventually develops either as a feeling of heaviness due to the greatly enlarged organ, or sharp pain reflecting capsular stretching or invasion. Weakness and weight loss may predominate and, once established, progress rapidly Sometimes persistent unexplained fever appears weeks in advance of any localizing manifestation; indeed, malignant disease of the liver must be given serious consideration in any case of prolonged obscure fever The organ itself shows progressive and rapid enlargement, often becoming tremendous Its surface is irregular or nodular, and eventually tender Jaundice occurs only when a major bile passage is obstructed, or, in the terminal stages, when virtually all of the liver is involved In the absence of portal, splenic, or hepatic vein obstruction by external pressure or intraluminal invasion, enlargement of the spleen is rare, an important point in differentiating between cancer and cirrhosis Ascites, from portal or hepatic vein obstruction, or from peritoneal implants is sometimes a late feature. Since the process is one of replacement rather than destruction of hepatic cells, the laboratory indications of parenchymal impairment, in contrast to hepatitis and cirrhosis, are not striking except when flow of bile is

seriously hampered, or when the patient lives long enough for virtually all of his liver substance to be replaced by tumor.

As a rule, x-ray is helpful only when it will detect the primary tumor In cancer of the pancreas, the hepatic metastases occasionally calcify and become evident radiologically as small ill-defined densities in the liver area.

Metastatic Sarcoma. Usually secondary to osteogenic sarcoma, this is marked by rapid enlargement of the liver, cachexia, and other systemic manifestations of malignant disease

Lymphoma. The liver is moderately enlarged and sometimes nodular. As a rule, symptoms are related to the disease as a whole Those referable to the liver are not striking except when obstructive phenomena are created, as for example, by pressure on the common or hepatic duct by an enlarged lymph node

THE GALLBLADDER AND BILE DUCTS

Symptoms and signs directing attention to the gallbladder and bile passages are

Biliary Colic. *Stone in the gallbladder or a major duct produces a characteristic type of pain which begins suddenly, especially at night or shortly after a full meal, is sharp, cutting, or knife-like, and rapidly increases in intensity,* causing the patient to double over and sweat profusely The pain does not occur spasmodically or in waves, strictly speaking, therefore, the term biliary colic is incorrect, but we continue to use it since it is so generally accepted. It is felt in the epigastrium or right upper quadrant Often it is referred to the right scapular region or occasionally into the upper chest or neck or, rarely, into the right lower quadrant, left upper quadrant or down the right arm. Associated nausea and vomiting are frequent, especially with a severe attack. Untreated, the pain will endure for a variable length of time, usually from one to several hours or longer. Tenderness just beneath the right costal margin and sometimes over the whole hepatic region accompanies it Between episodes there is no pain but the gastrointestinal disturbances described below may constantly or intermittently harass the patient. As a general rule, pain of the greatest severity, accompanied by nausea and vomiting, favors stone in the cystic or common duct, less acute distress, the gallbladder. Biliary colic is distinguished *from ureteral colic* by the tendency of the latter to originate in the flank, spread downward into the bladder region and genitalia, and show intermittent *variations of intensity From peristaltic pain, biliary colic is* differentiated by its greater severity, different location and lack of rhythmic fluctuation At times other causes of pain resembling biliary colic, such as *the gastric* crisis of tabes dorsalis and plumbism must be considered, and excluded by pertinent investigative procedures.

Pain resembling biliary colic can occur without stones in disease of the gallbladder or ducts and sometimes in liver disease, especially cirrhosis

Gastro-Intestinal Disturbances. Indigestion, flatulence, and constipation are common between acute episodes, nausea and vomiting during them

Jaundice. Posthepatic jaundice may result from obstruction of the hepatic

or common duct by stone or, in acute cholecystitis, by associated inflammatory edema In the ordinary attack of gallstone colic due to chronic cholecystitis with stones, jaundice does *not occur*

Tenderness. This is usually confined to a small area beneath the right costal margin mesial to the midclavicular line but may extend further to the right and across the epigastrium Sometimes it can be brought out only by pressing upward under the costal margin while the patient inspires

Muscle Spasm. Rigidity can be detected beneath the right costal margin when the gallbladder is acutely inflamed

Palpable Gallbladder. *The gallbladder is palpable only when enlarged and* tense. It then presents as a smooth, rounded, pear-shaped mass extending from beneath the right costal margin near midclavicular line, downward toward the umbilicus. It is sometimes confused with a large right kidney or a right upper quadrant tumor. The common causes of enlargement are

1 Stone in cystic duct The gallbladder becomes distended by retained secretion due to impaired drainage (*hydrops*), but in the presence of associated acute *cholecystitis, tenderness and spasm may so hamper palpation that the enlargement* is undetectable Secondary infection may render the retained secretion purulent (*empyema of gallbladder*) Jaundice may or may not be present

2 Carcinoma of head of pancreas, ampulla of Vater, or common duct, or obstruction from without the duct, as by an enlarged metastatic or lymphomatous lymphnode Jaundice, usually intense and progressive, is the rule

Stone in the common duct does not customarily cause an enlarged gallbladder *(see* Courvoisier's law, *above)*

Systemic Signs of Infection These occur with any acute inflammatory process

Cholelithiasis

Although encountered particularly after the age of 40, this disease is found in younger persons more frequently than is generally supposed *It predominates* in females Some patients harbor gallstones for years without any indications of ill health; others complain of constant or periodic indigestion, flatulence, and epigastric distress which are often aggravated or precipitated by a large meal or over-indulgence in certain foods, especially those which are fried or high in roughage. Sooner or later, attacks of biliary colic are likely to develop, they may last from a few minutes to hours and may recur at intervals of a week or so, *or no oftener than every three to* four years or longer The pain is attended by acute tenderness over the gallbladder region and followed by residual soreness lasting several days Local spasm, fever and leukocytosis due to associated inflammation of the gallbladder may accompany the acute episode Jaundice is present only when there is accompanying obstruction of a major duct by stone or inflammation. *It is sometimes clinically* impossible to determine the site of a stone but the following considerations may lead to the correct diagnosis.

Stone in Gallbladder. Enlargement and jaundice are not usual but may occur if the gallbladder becomes acutely inflamed

Stone in Cystic Duct. Stone here may remain latent and virtually asympto-

matic for a considerable period, or create symptoms comparable to and perhaps reflecting associated chronic cholecystitis. Impaired drainage may create hydrops often sufficient to make the gallbladder palpable but in the long-standing case, thickening and fibrosis are likely to prevent enlargement even in the presence of block. Since bile flow is not impaired, jaundice is not a feature. An attack of superimposed acute cholecystitis with intense pain, fever, tenderness, and perhaps jaundice, is possible at any time.

Stone in Common Duct. As a rule, the calculus lodges in the ampulla of Vater and characteristically sets up the so-called ball-valve action, creating intermittent episodes of obstruction to bile flow. Recurrent attacks of pain, jaundice, fever and leukocytosis lasting for a few days to a week, are the rule. During the episode the pain, although not definitely colicky, tends to vary in intensity. Common duct stone is always suggested by such attacks if they recur repeatedly during a period of weeks or months. The gallbladder is not enlarged. Sometimes jaundice is not evident Between episodes, the patient may be symptom-free It is always possible for a stone to become lodged in such a way as to create *continuous obstruction*, whereupon jaundice and evidence of infection persist. The long-standing case is likely to be complicated by eventual development of low-grade cholangitis and obstructive cirrhosis.

To differentiate between obstruction due to common duct calculus and tumor as a cause of obstruction is difficult When stone is at fault, one expects a history suggesting gallbladder trouble in the past. Jaundice is likely to wax and wane, fever and leukocytosis are present, and the gallbladder cannot be felt Clumps of calcium or sodium bilirubinate or cholesterol crystals in material obtained by duodenal drainage are suggestive. In obstruction due to tumor, jaundice is likely to become progressively more intense, the gallbladder becomes palpable,



FIG 31 7 A Plain film of abdomen showing a cluster of radiopaque faceted stones in gallbladder B Oral cholecystogram showing filling defects due to presence of large non radiopaque stones.



FIG 31 8 Intravenous cholangiogram Common bile duct outlined by contrast medium Arrows point to filling defects due to non radiopaque stones

and fever and leukocytosis are not prominent *Often positive diagnosis can be made only by surgical exploration.* Even at operation dense inflammatory tissue surrounding the ampulla in a case of stone, may be mistaken for cancer Rarely, one may find stenosis of the duct or sclerosing cholangitis instead of stone or tumor

X-ray will almost always demonstrate stone in the gallbladder A radiopaque concretion will be evident on a scout film but calculi are so often non-radiopaque that, as a rule, one must rely on demonstration of one or more filling defects in a cholecystogram Because filling defects may be obscured by surrounding dye when the patient is prone, views must be taken while he is erect or (with rays directed horizontally) in the right lateral decubitus, in these positions stones will become dependent or float as a layer in the medium, whereupon the filling defects they create will usually but not always be apparent. A normal oral cholecystogram does not exclude stone in a major duct. Cholecystitis, which usually predicates calculus disease, is sometimes reflected by poor to absent filling but here one should not rely on a single routine examination; disease can be assumed only if impaired filling is repeatedly demonstrated.

If oral cholecystograms are non-definitive, intravenous cholangiography should be tried In a high percentage of cases this procedure will outline the

gallbladder and ducts and, even when the gallbladder has been removed, the hepatic and common ducts. It is especially valuable in the detection of cholecystitis, and stone or other partial obstructive lesion of a major duct. Neither the oral nor intravenous method is dependable in the presence of an appreciable degree of intra- or posthepatic jaundice or, even though jaundice is absent, in severe disease of liver parenchyma.

Cholecystitis

Acute. Acute cholecystitis may be suppurative or non-suppurative. It is almost invariably associated with a stone in the cystic ampulla or duct. The symptoms may be mild and indistinguishable from those attributable to chronic cholecystitis during an attack of colic. The more severe form causes intense pain accompanied by tenderness and local rigidity, nausea, vomiting, fever, and leukocytosis. Jaundice is rare but occasionally occurs as a result of impaired bile flow from associated inflammatory edema of common or hepatic duct. A mass due to hydrops or empyema of gallbladder, or to inflammatory reaction of surrounding tissue, may be felt beneath the costal margin. Occasionally a scout film will show a faint outline of the enlarged gallbladder; since the diseased organ is unlikely to concentrate the dye, contrast studies are rarely helpful Perforation followed by shock and peritonitis is a possibility in the severe case

Chronic. Chronic cholecystitis is also almost always associated with calculus disease It may be asymptomatic for years or marked by periodic or continuous indigestion, epigastric distress, flatulence, and gaseous eructations. Jaundice is not present Acute episodes of pain, tenderness, and perhaps local spasm usually with nausea and vomiting may occur at varying intervals, sometimes no oftener than once in three to four years or longer. Diagnosis must be confirmed by cholecystographic demonstration of stones or, on several examinations, faint or absent concentration of the opaque medium

Rarely, cholesterosis of the gallbladder creates a similar clinical picture. Here the cholecystogram is likely to be normal but the diagnosis may be confirmed by demonstration of cholesterol crystals in material obtained by duodenal drainage

Cases presenting abdominal discomfort, flatulence, and other mild digestive complaints are too often carelessly diagnosed as chronic cholecystitis and subjected to unnecessary surgery This diagnosis should not be made unless one can obtain a typical history, preferably one of biliary colic and/or evidence of trouble by cholecystography or duodenal drainage.

Cholangitis

Acute. Usually developing as a complication of cholelithiasis, occasionally of a severe acute infection, such as pneumonia or typhoid fever, this is a suppura-

DISEASE	REGIONAL TENDERNESS	SIZE OF LIVER	JAUNDICE	SIZE OF SPLEEN
1 Passive congestion	Slight to moderate	Enlarged Occasionally small in late stage of prolonged case	Absent Possibly slight in late stage	Normal or slightly enlarged Rarely palpable
2. *Acute hepatitis (mild to moderate).*	*Slight to moderate*	*Slightly to moderately enlarged*	*Present (occasionally subclinical)*	*Normal or slightly enlarged*
3 Acute hepatitis (severe)	Slight to severe.	Diminishes as process progresses May become very small	Marked	Slightly enlarged
4 Chronic hepatitis	Absent to moderate	Enlarged Increases during exacerbation	Absent to slight	Normal or slightly enlarged
5. *Multiple abscesses of liver*	*Slight to moderate* Possibly only compression tenderness	*Slightly enlarged*	*See remarks*	*Normal or slightly enlarged*
6. *Solitary abscess of liver*	*Slight to moderate* Possibly only compression tenderness	*Enlarged, usually upward*	*Rare*	*Normal*
7 Alcoholic or post-necrotic cirrhosis (early)	Slight to moderate Occasionally absent	Enlarged	Variable	Slightly enlarged
8 Alcoholic or post-necrotic cirrhosis (late)	Absent	Usually somewhat enlarged, occasionally small in alcoholic Small in post-necrotic	Slight	Slightly to moderately enlarged
9 Obstructive and chol*angiolitic cirrhosis*	Slight to moderate	Moderately to greatly enlarged	Chronic, fluctuating from slight to moderate	Moderately enlarged
10 Syphilitic cirrhosis	Absent	Normal to moderately enlarged	Absent	Normal to slightly enlarged

(Continued pp

ASCITES	SIZE OF GALL-BLADDER	URINE (BILIRUBIN TEST)	COLOR OF STOOLS*	REMARKS
Present or absent	Normal	Negative	*Normal*	
Absent	Normal	Positive	Normal to light	
Frequently present	Normal	Positive	Light	
Absent	Normal	Negative. Perhaps slightly positive during exacerbation	Normal	Manifestations vary from time to time
Usually absent	See remarks	Negative or slightly positive	Usually normal	Jaundice absent or slight, gallbladder normal with pylephlebitis or generalized infectious process. Jaundice prominent and gallbladder possibly enlarged with acute suppurative cholangitis. Septic fever and leukocytosis present
Absent	Normal	Negative	Normal	Usually associated with amebiasis, rarely with pylephlebitis. Septic fever and leukocytosis present
Absent. Occasionally slight	Normal	Negative or slightly positive	Normal	
Typically present	Normal	Negative or slightly positive	Normal	Esophageal varices common. Rupture of varices may cause death before ascites appears
Present in terminal stage only	Normal	Intermittently negative and positive, depending on degree of jaundice	Normal. May be light when jaundice is pronounced	Often history of obstruction in biliary tract, rarely of previous hepatitis. Course of disease marked by remissions and exacerbations
Usually absent	Normal	Negative	Normal	Very irregular edge (*hepar lobatum*). Positive serologic test

610–612)

DISEASE	REGIONAL TENDERNESS	SIZE OF LIVER	JAUNDICE	SIZE OF SPLEEN
11. Pigment cirrhosis	Absent	Enlarged. Possibly small *in late stage*	Absent (bronze discoloration of skin due to iron pigment)	Normal or slightly enlarged
12. *Metastatic carcinoma* of liver	*Present in late stage*	Enlarges progressively, sometimes reaching huge size	Absent. Occasionally present	Normal
13. Chronic cholecystitis with cholelithiasis	Moderate to severe with colic Otherwise absent	Normal	Absent	Normal
14. Stone in cystic duct	Severe during acute attack	Normal	May be present during acute attack	Normal
15. Stone in common duct	Moderate with severe colic	Usually normal	Intermittent	Usually normal
16 Carcinoma of head *of pancreas, ampulla* of Vater, or common duct	Absent until late stage	Enlarges progressively *due to obstruction* and/or metastases	Pronounced and progressive. May be intermittent in early cancer of ampulla	Normal

* Only the effect of the presence or absence of bile pigments and undigested fats is considered Other factors affect

Fig 31 9 Chart showing important clinical

of septic fever, chills, leukocytosis, and other signs of acute severe infection, *along with moderate jaundice and slight to moderate enlargement and tenderness of the liver* In a case of severe general infection, the disease is suggested by intensification of the systemic symptoms, tenderness and enlargement of the liver, and jaundice Unless the infection can be controlled by specific therapy, the course is rapidly downhill coincident with that of the underlying disease. In the late stage of the unsuccessfully treated case, multiple small abscesses of the liver are likely

Chronic. This usually develops gradually as a result of stasis and infection in common duct obstruction, or of infectious hepatitis The picture is similar to that of obstructive or cholangiolitic cirrhosis.

The accompanying diagram (Fig 31 9) showing the distinctive clinical features of the more important diseases of the liver and biliary tract can be used as

ASCITES	SIZE OF GALL-BLADDER	URINE (BILIRUBIN TEST)	COLOR OF STOOLS*	REMARKS
Possibly present in late stage	Normal	Negative	Normal	Sugar may be found in urine. Iron pigment in skin may be demonstrable on pathologic examination before evident clinically. Elevated serum iron and iron binding capacity
Possibly present in late stage	Normal	Negative. Positive if jaundice present	Normal. Light with complete biliary obstruction	Jaundice depends on degree of block in intrahepatic or extrahepatic ducts. Ascites, if present, usually due to peritoneal implants. Course rapidly downhill. Fever usually present
Absent	Normal or small	Negative	Normal	Stones may be present in gallbladder for years without giving symptoms. With acute attack of colic, signs of acute cholecystitis often appear
Absent	Usually slightly or moderately enlarged	Negative unless jaundice occurs	Normal unless jaundice pronounced	
Absent	Normal or small	Intermittently positive	Intermittently normal and light	Usually characterized by recurrent attacks of pain, fever and jaundice with intervals of freedom from symptoms. In long-standing case, picture of obstructive cirrhosis may develop
Rare. Occurs occasionally from peritoneal implants or portal obstruction	Usually enlarged	Strongly positive	Light	Rapid downhill course

ing color of stools, such as blood, are disregarded

findings in various diseases of liver and biliary tract

a guide, provided one bears in mind that exceptions to the usual rules are often encountered.

PANCREAS

The diagnosis of pancreatic disease is often missed because of failure to think of this structure as a possible site of trouble, its inaccessibility to physical examination, the limitations of x-ray studies, and the similarity of pancreatic symptoms to those originating in near-by structures, especially the biliary tract, stomach, and heart. Midline epigastric pain or tenderness introduces the possibility of pancreatitis or tumor but other more likely causes must always be considered. A palpable mass suggests tumor or cyst but here too some other organ may be at fault. The likelihood of pancreatic disease is strengthened by discovery of sugar in the urine, or (provided they cannot be accounted for by some

other cause such as jaundice, diarrhea, or ingestion of large amounts of meat, fat, or oily preparations) passage of copious soapy stools or repeated detection in the feces of excess muscle fibers or neutral fats. But absence of these findings by no means excludes the pancreas, since its reserve is so great that a large portion of it can be destroyed without functional impairment.

Pancreatitis

Acute. This often occurs independently. Frequently, however, it is encountered in the presence of biliary tract disease, especially stone in the common duct, the latter may have been hitherto asymptomatic. Penetration of a duodenal ulcer is an occasional cause The typical attack begins abruptly with intense epigastric pain, nausea and vomiting; often it starts after an unusually large meal or following generous ingestion of alcohol The pain is persistent, severe, deep, rarely it is colicky or intermittent, and often extends through to mid-back or left costovertebral region Relief from an opiate or other potent analgesic is rarely complete, the patient will continue to complain of some epigastric or back distress The pain is easy to confuse with that of acute cholecystitis, perforated peptic ulcer, acute intestinal obstruction, myocardial infarction, and mesenteric thrombosis Obstipation results from associated paralytic ileus Tenderness is pronounced in the epigastrium, on deep palpation it may be traced into the left hypochondrium Sometimes it is also found at the left costovertebral angle Epigastric spasm may or may not be striking, rarely one can feel fulness or an indefinite mass Fever is moderate to high but may be normal in the presence of shock The leukocyte count is elevated, often to an unusually high level. The patient's condition is often alarming from the start Early peripheral circulatory collapse is likely Some cases recover within a few days, some die after a rapid downhill course In others, symptoms subside somewhat within the first two or three days but moderate pain, tenderness, spasm, fever, and leukocytosis persist Discharge of bile, pancreatic secretion, or blood from the necrosed organ will cause diffuse abdominal tenderness most pronounced along the peritoneal gutters Slight jaundice may appear Some of these cases gradually recover Pancreatic pseudocyst may be the end-result (*see below*)

In early acute pancreatitis the amylase content of the blood is markedly elevated, an important point in differentiating this disease from other upper abdominal catastrophies After a day or so, it returns to normal or lower as a result of recovery or total impairment of the organ Pronounced depression of serum calcium is another helpful sign

X-ray shows paralytic ileus Sometimes one's premise that the pancreas is the site of the trouble can be materially strengthened by demonstration of a pressure defect or widening of the duodenal loop, perhaps with associated coarsening of its mucosal folds When the pancreas is unusually swollen, a pressure defect of the stomach may be evident

Although the picture just described represents the typical case of severe pancreatitis, it may well be that the disease occurs in a milder form much more frequently than is generally supposed It is quite possible that lesser short-lived



Fig 31 10 Acute pancreatitis Lumen of second portion of duodenum decidedly narrowed by external pressure on its medial wall from swollen pancreas A Antrum of stomach B First portion of duodenum C Arrows point to narrowed second portion of duodenum (A comparable picture can be created by carcinoma involving head of pancreas)

attacks of epigastric pain and tenderness, nausea and vomiting attributed to improper eating, food poisoning, indigestion or even gallstones actually reflect transient low-grade *acute pancreatitis*

Mild to moderately severe pancreatitis is an occasional complication of mumps

Chronic This is a progressive disease characterized at first by repeated episodes of acute pancreatitis (*chronic relapsing pancreatitis*) which eventually appear to lead to severe fibrosis, perhaps with calcification, and resultant persistently impaired function During the earlier phases, the patient experiences recurrent mild to severe episodes of acute trouble as described above As time goes on, these are less prominent but pain and digestive difficulties become virtually continuous, with perhaps periods of accentuation Eventually diarrhea, steatorrhea with large, bulky stools, weight loss and other indications of malnutrition dominate the picture Frank diabetes or a diabetic-type glucose tolerance curve may develop Depending on the degree of parenchymal destruction, blood amylase is normal or low The stools are profuse, bulky, and often, microscopically, show fat and the typical square-ended, clearly striated fibers of undigested meat If calcification occurs, x-ray will show calcific mottling in the pancreatic area, sometimes this is discovered in the absence of clinical indications of trouble In the occasional case, radiographic changes similar to those of acute pancreatitis are seen

FIBROCYSTIC PANCREATITIS. This is a disease of unknown cause encountered in infants and children It is marked by steatorrhea, malnutrition, and evidence of progressive bronchopulmonary disease.

TUMOR OF PANCREAS

Carcinoma. In accordance with accepted terminology, we apply the term *carcinoma of pancreas* solely to cancer arising in the acinous system. Carcinoma of the *islands of Langerhans*, which is rare, is designated as such or as *islet-cell* carcinoma

Cancer of the pancreas is rarely diagnosed early because its symptoms at this stage—indigestion, flatulence, anorexia, and epigastric pain—are often so nonspecific. At times, however, the single complaint of persistent boring epigastric pain perhaps extending to the back is striking. Absence of objective manifestations of trouble frequently leads to a mistaken diagnosis of psychoneurosis Pancreatic cancer must always be seriously considered in the presence of the above symptoms, especially when they develop in midlife or beyond, and when one cannot prove by x-ray and other studies the existence of disease in some near-by structure, such as cholecystitis, gastric carcinoma, or peptic ulcer Under these circumstances one is occasionally justified in recommending surgical exploration in the hope of establishing a diagnosis and perhaps finding a removable lesion. Certain features of the clinical picture are related to the part involved.

Cancer of the *head* is marked by a pressing, aching or boring pain deep in the epigastrium, often extending to the right hypochondrium and sometimes even to the right scapular area Progressive jaundice due to obstruction of the common duct is the rule, but, contrary to usual teaching, is the presenting symptom in not over one-quarter of cases; pain, anorexia, and pronounced weight loss are more likely to appear earlier. Rarely, painless jaundice is the initial manifestation. Intractable itching may precede clinical icterus by weeks or longer With obstruction, progressive enlargement of the gallbladder and liver is the rule. In a thin or emaciated person, the tumor itself may be palpable The spleen is not enlarged, in the jaundiced person absence of splenic enlargement favors biliary obstruction due to tumor or other cause, as opposed to intrahepatic disease.

Cancer of the *body* or *tail* creates intense, constant, or intermittent, deep boring pain in the epigastrium and to its left, and in the back If the body is involved, the back pain is felt in midline at the level of the twelfth thoracic or first lumbar vertebra, if the tail is involved, toward the left costovertebral angle Often the discomfort, especially that in the back, is increased by lying supine and somewhat relieved by lying prone or by sitting with the trunk bent forward With time, pain spreads to the periumbilical and adjacent areas presumably from invasion of the celiac plexus Jaundice and other signs of common duct obstruction are not a feature; the spleen may become enlarged by invasion and obstruction of its vein

Irrespective of its site, well-established cancer of the pancreas is noteworthy

for its rapid downhill course with swiftly developing loss of weight, strength, and appetite Local invasion of duodenum may give rise to melena; invasion of portal vein, or its obstruction by enlarged regional nodes, to ascites and dependent edema. Ascites may also be created by peritoneal implants Widespread lymphatic or blood stream metastasis is usual unless the patient dies of inanition beforehand Indications of pancreatic insufficiency are rare although in the late stages, glycosuria or large soapy stools may be observed.

Spontaneously developing thrombophlebitis of the veins in the lower extremities or elsewhere is common in carcinoma of the pancreas Sometimes it precedes any other indication of the disease so that when thrombophlebitis is encountered in the absence of one of its usual causes such as immobilization, stasis, or trauma, the possibility of early pancreatic carcinoma must always be entertained.

When the head is involved, x-ray may demonstrate local pressure on the medial aspect of the duodenum, generalized widening of its loop or, in the late stages, loss of mucosal pattern of duodenum or fundus of stomach resulting from invasion In cancer of the body or tail, x-ray is rarely helpful, although occasionally there will be evidence of pressure on the body of the stomach

Islet-Cell Adenoma. This rare disease deserves mention because of the hyperinsulinism which it can create A single tumor is the rule The patient is subject to repeated attacks of hypoglycemia which, with time, become increasingly severe and frequent Obviously, they are most likely to occur in the fasting state—in the early morning or shortly before meals—and can sometimes be aborted by anticipatory feedings The mild attack is characterized by weakness, nervousness, faintness, tremulousness, anxiety, and sweating, the more severe attack, by confusion, aphasia, manic behavior, convulsions, and unconsciousness Between episodes the patient feels well The case is often mistakenly diagnosed anxiety or hysteria If he has learned that attacks can be aborted by frequent eating, the patient may be overweight when first seen. Fasting blood sugar is low and a specimen taken during an attack, excessively so The glucose tolerance test may be normal or show a low, flat curve Insulin tolerance test shows low blood sugar values throughout Very rarely a similar picture is created by multiple pancreatic adenomas associated with parathyroid and pituitary adenomas and, in children, by diffuse hyperplasia of the islands of Langerhans

Islet-Cell Carcinoma This is also rare It is a rapidly growing tumor with widespread metastasis Hypoglycemia may or may not occur. Diagnosis is difficult, it may be made by discovery and biopsy of a metastatic lesion

Cyst of Pancreas

True Cyst. This is rare. Depending on its cause, it may be single or multiple True cyst is lined with epithelium, although when examined microscopically, the epithelial layer may not be evident, owing to destruction by pressure from retained secretion It may be congenital (fibrocystic pancreatitis), embryonal (dermoid or teratoma), or neoplastic (cystadenoma or cystadenocarcinoma). One

or more retention cysts may occur from obstruction of a duct by *stone, tumor,* or the inflammatory changes of chronic pancreatitis. Very rarely, cysts are found in certain parasitic diseases such as echinococciasis, ascariasis, and taeniasis. The *clinical picture* is likely to be a reflection of the underlying disease. Symptoms and signs referable to a cyst itself are usually insignificant unless it becomes large enough to create indications of pressure on an adjacent structure, be evident as a mass, or destroy a large amount of pancreatic tissue.

Pseudocyst. As a rule, pseudocyst is a sequela of an attack of acute pancreatitis or injury to the pancreas from an abdominal blow. Fluid from hemorrhage and tissue necrosis becomes walled off within the organ, or more likely extends into and becomes encapsulated in the lesser peritoneal cavity. *In contrast* to true cyst, the wall is of inflammatory origin and not lined with epithelium. The manifestations vary with the acuteness of the process, the size of the cyst, *and its rapidity of enlargement. The less acute or more slowly developing lesion* may cause no symptoms, perhaps be attended by indigestion, flatulence, and mild epigastric pain, or first become evident to the patient or his physician as fulness or a mass in the epigastrium. In the more acute or rapidly developing case, pain in the epigastrium occasionally referred to the back or left flank, sometimes anorexia, nausea, or vomiting, and perhaps systemic manifestations such as fever and loss of weight are likely. On physical examination one can usually discover a firm, rounded mass in the epigastrium or just to its left. Usually, because of the thickness of the fibrous wall, fluctuation characteristic of encapsulated fluid is not obtainable on palpation. In some cases, presumably as a result of intermittent discharge of retained fluid through the pancreatic duct, the cyst will vary in size from time to time. Laboratory studies are rarely helpful. Plain abdominal films show a rounded, well demarcated shadow of increased density in upper mid- or left abdomen; barium studies, a pressure defect, or displacement of stomach, duodenum, or bowel.

Diabetes Mellitus

In its mild form or in old persons, this may exist undetected for some time and is likely to be discovered on routine urine examination. A severe, acute case or a long-standing one usually shows some of the following: loss of weight and strength coincident with increased appetite, polydipsia, polyuria, and, often in females, itching of the vulva. Sometimes the first sign is diabetic coma, gangrene of a toe, the pain or paresthesia of diabetic neuritis, or disturbance of vision due to cataract or diabetic retinitis. *There are no physical signs related* to the pancreas itself. The diagnosis is confirmed by demonstration of persistent glycosuria and hyperglycemia and, in the doubtful case, a diabetic-type glucose tolerance curve.

In a case which is resistant to insulin therapy, hemochromatosis must be considered.

SPLEEN

Investigation of the splenic region is aimed chiefly at detecting increased size of the organ, a reflection of either primary disease or trouble elsewhere. Since

certain diagnostic possibilities are suggested by the degree of enlargement, one classifies it as slight, moderate, or great. No sharp line can be drawn but the following arbitrary criteria serve as a working rule:

Slight Enlargement. The tip is felt 1–3 cm. below the costal margin or becomes palpable just beneath it as the spleen descends with deep inspiration. As a rule, no local symptoms are evident. With any degree of enlargement, however, associated perisplenitis may create overlying discomfort, perhaps varying with respiration, and sometimes audible or palpable friction resembling pleural friction.

Moderate Enlargement. The tip lies from 3 cm. below costal margin to the level of the umbilicus. A sensation of fulness or heaviness in the left upper quadrant may be described.

Great Enlargement. The tip is below the level of the umbilicus. In addition to heaviness in the abdomen, the patient may be aware of regional prominence or swelling.

Palpation

The bimanual method is preferable. The technique can be mastered only by long practice, slight enlargement is often missed by the inexperienced. The patient lies on his back with thighs and legs partially flexed and heels resting on the table. Standing or sitting on the patient's right, the examiner places his left hand on the posterolateral aspect of the lower left thorax and upper abdomen. His right hand rests on the anterior abdominal surface parallel to the thoracic margin, with his fingers approximately between the left parasternal and anterior axillary lines. The left hand, exerting firm pressure against the lower ribs and upper loin, pulls this region toward the right hand and at the



FIG. 31 11. Bimanual palpation of spleen. Correct position of examiner's fingers, hands and arms.



FIG. 31 12 Incorrect palpation of spleen Examiner is too high above patient, his right hand is pressing too deeply into abdomen, his left hand is not in the loin but digging into the side Patient's thighs are not flexed

same time slides the skin and subcutaneous tissues anteriorly and medially The latter maneuver gives the fingers of the right hand a slack rather than taut segment of abdominal wall through which to feel. The patient is then instructed to breathe deeply through his mouth, as the end of inspiration is reached, the examiner presses his right fingers upward under the costal margin The fingers must be partially relaxed and only slight pressure exerted lest the spleen be forced backward or its inspiratory descent impeded Some observers prefer to palpate with the right fingers perpendicular to the costal border This is worth trying in the doubtful case Furthermore, slight enlargement not discovered with the patient supine can sometimes be detected if the same procedures are carried out with him in the right decubitus with thighs and legs flexed, or standing up with his trunk bent slightly forward

If the spleen is slightly enlarged, its tip should be felt just under the costal margin as it descends with inspiration If it is not felt at this point, the procedures described above should be repeated with the palpating fingers feeling for it at successively lower levels One seeks the tip because, in lesser degrees of enlargement, pressure on the anterior surface will force the spleen backward so that nothing unusual will be felt Obviously, this does not pertain in the case of a moderately or greatly enlarged organ which occupies so much space that it cannot be forced appreciably backward and will create, under the palpating fingers, a feeling of resistance suggestive of a mass.

Unimanual palpation should also be performed in the equivocal case The technique is essentially the same except that the examiner stands at the patient's left shoulder and feels for the tip first with his fingers hooked under the

costal margin and then, if indicated, pressed into the abdomen at lower levels If necessary, both the supine and right lateral positions should be tried.

When the spleen is soft, as in typhoid fever or other acute infection, it is more likely to be overlooked than when firm, as in lymphoma, hepatic cirrhosis, or leukemia. Downward displacement from such a cause as large pleural effusion or high pressure pneumothorax must be differentiated from slight enlargement.

A greatly enlarged spleen appears as a mass extending obliquely downward from the left costal margin toward the symphysis pubis Readily appreciated on either uni- or bimanual palpation, it occupies most of the left side of the abdomen and appears to lie just beneath the wall The latter may show regional prominence or bulging The tip is found at the level of the iliac crest or even lower. The organ retains its characteristic shape, is firm and smooth; its medial and lateral edges, sometimes quite sharp, can usually be outlined. One or more notches are often made out on the medial surface and, when found, help to identify the structure as spleen. When moderately or greatly enlarged, the latter is easy to confuse with a large kidney, intra-abdominal neoplasm, or mass of inflammatory tissue These, however, appear to lie deeper in the abdomen and, as a rule, have neither a clearly defined edge nor a palpable notch; a neoplastic or inflammatory mass is likely to be irregular and hard.

Percussion

When palpation fails to disclose suspected enlargement, percussion should be tried, but is rarely helpful. Very light strokes are essential. The zone of normal splenic dulness is 6–8 cm. in width and extends from the ninth to eleventh ribs in left mid- and anteroaxillary region, it is often obscured by gas in the stomach or bowel Slight splenomegaly alters these limits so little that appreciable change can be detected, if at all, only by a highly skilled observer One must, as a rule, rely on palpation and x ray If the organ is palpable, percussion is obviously unnecessary

X-ray Findings

If, for some reason, such as a fat, distended, or tensely held abdomen, enlargement of the spleen cannot be made out clinically, it may be demonstrated by x-ray In contrast to the liver, determination of splenic size by plain abdominal film is regarded as quite dependable Especially when it is enlarged, the shadow of the organ is well outlined Barium studies of stomach or bowel may disclose a pressure defect created by a moderately or greatly enlarged spleen. By determining the location of a shadow, they may also help one to decide whether a palpable structure is spleen, kidney, or intra-abdominal mass.

Causes of Splenic Enlargement

An enlarged spleen may be an important factor in confirming a diagnosis postulated on the basis of other evidence, or be the presenting sign which actuates further study and eventual diagnosis The blood or bone marrow picture,

biopsy of liver, a lymphnode or the spleen itself may solve the problem. Sometimes surgical exploration becomes necessary.

The causes of splenomegaly could be grouped as follows. (1) infection, (2) blood dyscrasia, (3) liver disease, (4) impaired circulation, (5) tumor, (6) disturbance of metabolic processes, (7) miscellaneous. However, since the size itself suggests definite diagnostic possibilities, classification based on the relationship between causes and size seems better for the purposes of this book. In this connection, three important facts must be kept in mind:

1. The demarcations between slight, moderate, and great enlargement are arbitrary and ill-defined

2 The patient may chance to be examined before the spleen has reached the size typical of his particular malady

3 Following severe hemorrhage, a large spleen may shrink for a few days and mislead one by being smaller than would be expected in the illnesses under consideration.

In addition to the more common causes of splenic enlargement, certain rare diseases are discussed in the following pages because splenomegaly is so important in their diagnosis

Slight Enlargement

Chronic Passive Congestion in Long-standing Congestive Failure. Except when cardiac cirrhosis of the liver has developed, splenomegaly is rarely sufficient to be made out clinically and is usually a *post-mortem* finding

Acute Malaria.

Typhoid Fever.

Brucellosis.

Subacute Bacterial Endocarditis. Enlargement, usually slight, occasionally moderate, may be the result of toxemia, infarction, or both Infarction is suggested by one or more attacks of sudden sharp, relatively short-lived pain in the left hypochondrium

Other Acute or Subacute Infections. In septicemia, miliary tuberculosis, histoplasmosis, perhaps early syphilis, and others, slight enlargement of the spleen is likely Rarely, splenomegaly due to tuberculosis occurs without evidence of the disease elsewhere

Connective Tissue Diseases. Especially in polyarteritis nodosa, disseminated lupus erythematosus, and Still s disease, and sometimes in adult rheumatoid arthritis, slight to moderate splenomegaly may be found

Thrombocytopenic Purpura. Associated features are hemorrhagic tendency and marked diminution or absence of thrombocytes in the circulating blood Usually the spleen is not large enough to be palpable

Thalassemia Minor. In persons born of stock originating in countries bordering the Mediterranean, slight splenic enlargement is often found in the absence of symptoms or signs of any trouble. These cases represent a mild form of Mediterranean anemia, a hereditary disease peculiar to this group Careful study should reveal mild microcytic, hypochromic anemia, poikilocytosis out of pro-

portion to the degree of anemia, "target cells" and, in contrast to congenital hemolytic anemia, decreased osmotic fragility of red cells

When found in a person who has lived in an endemic area, slight splenomegaly, otherwise unexplainable, can perhaps be attributed to some previous infection such as malaria, brucellosis, or kala azar.

Slight to Moderate Enlargement

Rickets or Other Deficiency State of Childhood. The splenomegaly is probably due to associated severe anemia resulting from infection or iron deficiency.

Acute or Chronic Hepatitis.

Cirrhosis of Liver.

Lymphoma and, rarely, Metastatic Carcinoma.

Infectious Mononucleosis. Other features are fever, *pharyngitis, tonsillitis,* perhaps stomatitis, generalized lymphnodopathy, and diminished, normal, or increased leukocyte count with presence of abnormal mononuclear cells Liver enlargement and jaundice, usually mild but sometimes severe, are not uncommon An evanescent morbilliform eruption may occur Diagnosis can be confirmed by finding the characteristic morphologic variants in the blood smear. In some cases, even though the clinical picture is typical, these blood changes, being transient, are found only after repeated examinations Increased titer of heterophil antibodies in the blood serum is common but its absence does not exclude the disease

Acute or Chronic Leukemia. In contrast to other forms, well-established chronic myelogenous leukemia creates a huge spleen.

Pernicious Anemia. Before the advent of present-day therapy, splenomegaly was found in about 40 per cent of patients. Now it is encountered only in the long-standing untreated case and will disappear following adequate therapy.

Polycythemia Vera.

Hemolytic Anemia. In *congenital* hemolytic anemia, a hereditary disorder, one finds persistent mild jaundice and anemia, with occasional crises in which both become more pronounced. The spleen is moderately enlarged, the liver slightly or not at all The urine shows increased urobilinogen but no bilirubin The stools retain normal color and also show increased urobilinogen. The blood findings are normochromic, normocytic or simple microcytic anemia, spherocytosis, elevated reticulocyte count, and increased osmotic fragility Gallstones are a common complication and may confuse the picture by creating biliary obstruction and consequent posthepatic jaundice.

The *acquired* form is usually idiopathic, but sometimes associated with some other disease, such as malignant tumor, primary atypical pneumonia, or lupus erythematosus As a rule, reticulocytes, spherocytes, and increased fragility are less striking than in the congenital type Crises are rare The Coombs test is positive in most cases

Erythroblastosis Fetalis. Anti-Rh agglutinins, *developing in the mother's* blood and passing into the infant's circulation, destroy the fetal red cells and result in extensive compensatory overdevelopment of erythropoietic elements in

the marrow, liver and spleen Jaundice and anemia appear at birth or within a few hours. The spleen and liver are readily palpable; a bleeding tendency may be evident. Impaired cerebral nutrition causes early poor feeding, lethargy, perhaps spasms and rigidity. The blood shows macrocytic, normochromic anemia, nucleated red cells, reticulocytosis, immature white blood cells, and diminished thrombocytes. Death is the rule but prompt exchange-transfusions may be life-saving.

Sickle Cell Anemia. Almost entirely limited to the Negro race, this is a familial hemolytic disease marked by chronic mild jaundice, weakness, and anemia. The patient is subject to repeated crises of increased anemia and weakness, fever, arthralgia, and violent abdominal pains which may occur independently or in any combination In earlier years the spleen is enlarged but usually shrinks with time, and ends up as a small shriveled mass in which spotty calcification may be seen by x-ray. Chronic ulcers of the legs are a common feature. The blood shows normo- or macrocytic, hypochromic anemia with characteristic sickle, oval, or cigar-shaped red cells in the stained smear. If, as sometimes happens, these typical changes are not evident on a routine smear, they may be brought out by special techniques.

Splenic Abscess or Infarction.

Sarcoidosis. Slight to moderate splenic enlargement may or may not be found in association with other manifestations of the disease (*see* Chap. 27) Sometimes it is the only indication of trouble

Amyloidosis.

Schistosomiasis. In the early stages of *S japonicum* or *S. mansoni* infestation, splenic enlargement is presumably a response to deposition of eggs. Later, if the liver becomes cirrhotic, congestion secondary to portal hypertension is superimposed.

Moderate to Great Enlargement

Chronic Myeloid Leukemia. In the long-standing case, the spleen becomes enormous. This disease is the commonest cause of huge spleen in this country. Other, but less likely, possibilities are myeloid metaplasia, Gaucher's disease, and long-standing portal hypertension.

Myeloid Metaplasia. This is marked by moderate to massive splenomegaly, slight to moderate hepatomegaly, and hemorrhagic tendency The blood shows varying degrees of anemia with marked anisocytosis and poikilocytosis, nucleated red cells and immature granulocytes The bone marrow is often the site of fibrosis and sclerosis (*osteosclerotic anemia*), by x-ray the bones will show generalized increased density of cortex As the disease progresses, the patient may develop uricemia, with recurrent attacks of acute gout

In infants a rare but comparable disease occurs in a congenital form (*Albers-Schonberg* or marble bone disease)

G[illegible] Di[illegible]se. A chronic heredo-familial disease of disordered lipoid

[illegible]

erately enlarged liver, and changes in long bones demonstrable by x-ray are typical. Lymphnode enlargement is occasionally encountered. Pain in the extremities due to bone involvement may lead to a mistaken diagnosis of arthritis. Gingivitis with bleeding and soft tissue destruction, loss of alveolar bone, and loosening of teeth are likely. Especially characteristic are *pingueculae*—yellowish, wedge-shaped deposits in the sclerae extending from the cornea to the inner and outer canthi—and a yellowish tinge of the skin. The blood often shows anemia, leukopenia, and thrombocytopenia. By x-ray the most common finding is widening of the lower ends of the femurs producing the so-called *Erlenmeyer flask* deformity; areas of bone destruction, especially in the head of a femur or humerus, may also be observed. Diagnosis can be confirmed by bone marrow or splenic biopsy.

Niemann-Pick's Disease. Also due to disordered lipoid embolism, this disease is confined to infants. It is marked by tremendous enlargement of liver and spleen, hypochromic anemia, rapid mental and physical deterioration, and death within a few months to two or three years.

Thalassemia Major (Cooley's Erythroblastic Anemia). This is the severe form of Mediterranean anemia. It is marked by mongoloid facies, a tremendous spleen, slight to moderate hepatomegaly, and a typical blood picture—moderately severe anemia with microcytic, hypochromic, anisocytic, and poikilocytic red cells, large numbers of nucleated red cells, "target cells," and leukocytosis. Red cell fragility is diminished. Slight jaundice with increased urobilinogen in urine and stools is the rule. X-rays show medullary widening and cortical thinning of long bones, marked thickening of the diploe of the skull with perpendicular striation between its tables. In addition, the short bones show increased medullary trabeculation with a resultant mosaic pattern. Death usually ensues before puberty.

Chronic Malaria.

Congenital Syphilis.

Kala Azar (Leishmaniasis). Seen chiefly in parts of China, India and the Mediterranean area, this disease is initiated by malaise, and other nonspecific symptoms, intermittent or persistent fever, slight enlargement of spleen, diminished leukocyte count with granulocytopenia, and mild anemia. Later one finds severe prostration, wasting, moderate to greatly enlarged spleen, somewhat enlarged liver, well-established hypochromic anemia, and pronounced leukopenia. Double or even triple rises in temperature during 24-hour periods are significant, but not always present. Diagnosis is best confirmed by recovery of the organism from splenic pulp or bone marrow.

[illegible] roup of cases [illegible], leukopenia, [illegible] esophageal or gastric hemorrhage. They have been variously called *Banti's disease*, *Banti's syndrome*, *splenic anemia*, and *splenomegaly with early gastric hemorrhage.*

Obstruction to portal circulation *distal to the liver* accounts for many of the cases so designated. Splenic or portal venous flow may be impaired by healed

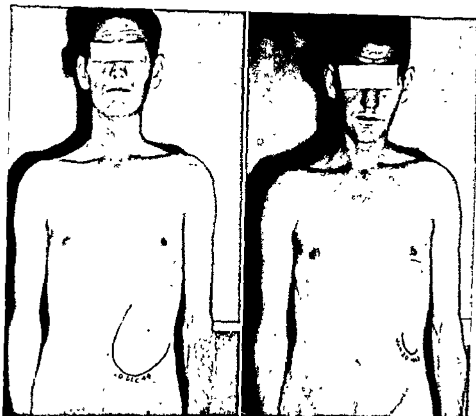


Fig 31 13 Kala azar A Pronounced enlargement of spleen B After one month of treatment Spleen decidedly smaller

thrombophlebitis, pressure from without by tumor or contracted scar tissue, or intraluminal tumor growth, as for example, an extension of cancer of the tail of the pancreas into the splenic vein When found in children, portal system thrombophlebitis is probably the end result of infection by way of the umbilical vein, occurring shortly after birth In any of these situations, progressive enlargement of the spleen is created by its impaired outflow Collateral *circulation develops to shunt blood around the block, hemorrhage results from* rupture of a gastric or esophageal varix in the auxiliary circuit Since the lesion is below the liver, indications of hepatic disease are not present Ascites may occur if the portal vein is involved but not if the obstruction is limited to the splenic branch

Obstruction *within the liver* is usually the result of cirrhosis, especially the portal or schistosomiasitic type Here one finds, in addition to the signs just mentioned, a large or small liver, ascites, and laboratory evidence of impaired hepatic function As a rule, the spleen does not become as large as in the group described above, perhaps because liver impairment contributes to earlier death It is now believed by most authorities that previous teaching concerning the second and third stages of so called Banti's disease was incorrect It seems more likely that when enlargement or other indication of liver disease is encoun-

tered, the case is actually one of intrinsic liver disease or some disturbance higher up.

Portal hypertension due to trouble *proximal to the liver* can be caused by thrombophlebitis, tumor invasion, or other obstruction of the hepatic vein (*Chiari's syndrome*), or by seriously impaired general circulation as in constrictive pericarditis or long-standing right heart failure. In the latter group, enlargement of the spleen, rarely pronounced, is a reflection of generalized venous stasis. In Chiari's syndrome, one finds a greatly enlarged, tender liver, enlarged spleen, rapidly developing ascites, and rapid decline. Here again, the process is too swift for the spleen to become greatly enlarged.

Cyst of Spleen. This is rare. One finds a large left upper quadrant tumor which extends downward and to the right, perhaps as far as the symphysis pubis. Its cystic nature is suggested by a sensation of resiliency obtained on bimanual palpation; sometimes a fluid wave can be detected. It is possible for the cyst to extend backward and upward so that the mass, although plainly evident on x-ray studies, is not striking on physical examination.

The accompanying chart (Fig. 31 11) shows the usual clinical findings in some of the more common diseases in which splenic enlargement is an important feature.

REFERENCE

1 Watson, C. J. Some Observations on the Recognition and Treatment of Commoner Forms of Hepatic Cirrhosis. Minnesota Med 35, 125, 1952

DISEASE	SIZE OF SPLEEN	SIZE OF LIVER	JAUNDICE	RED BLOOD CELL COUNT	WHITE BLOOD CELL COUNT	PLATELET COUNT	REMARKS
1. Pernicious *anemia*	Normal to *moderately* enlarged (*see text*)	Normal or slightly enlarged	Absent or very slight. Rarely deep	Decreased	Decreased	Normal or decreased	Jaundice may not be recognizable clinically but be demonstrable by blood bilirubin test. Red cells macrocytic Bone marrow biopsy may be necessary
2. Acute leukemia	Slightly to moderately enlarged	Normal or slightly enlarged	Absent	Decreased	Increased, normal or decreased	Decreased	Character of white cells usually establishes diagnosis. Bone marrow or lymphnode biopsy may be necessary
3. Chronic leukemia	Moderately to greatly enlarged	Moderately to greatly enlarged, occasionally slightly	Absent	Decreased	Increased, normal or *decreased* depending on phase	Increased, normal or *decreased*	Character of white cells usually establishes diagnosis *Bone* marrow or lymph node biopsy may be necessary
4. Lymphoma	*Slightly to* moderately enlarged	*Normal* to moderately enlarged	*Usually* absent	Normal Decreased in late stage	Usually normal May be slightly increased or decreased	Usually normal May be increased or decreased	Lymphnode, spleen or liver biopsy necessary
5. Infectious *mononucleosis*	Normal to *slightly* enlarged	Normal to slightly enlarged	Absent Occasionally slight to moderate	Normal	Slightly to moderately increased Occasionally *normal* or decreased	Normal	Differential count shows presence of abnormal monocytic or *lymphocytic* cells *Heterophil* reaction may be positive
6 Thrombocytopenic purpura	Normal to slightly enlarged	Normal	Absent	Normal to *greatly* decreased	Normal	Markedly decreased	Bone marrow biopsy helpful. Megakaryocytes normal to increased with decreased platelet production
7. Polycythemia vera	Slightly to *moderately* enlarged	Normal to slightly *enlarged*	Absent	Greatly increased	Slightly to greatly increased	Normal or slightly increased	Same picture occasionally seen in polycythemia secondary to congenital heart disease with cyanosis and in severe chronic cor pulmonale

8 Chronic Hemolytic Jaundice	Slightly to moderately enlarged	Slightly enlarged	Mild to moderate, and fluctuant	Slightly to moderately decreased	Normal	Normal	Increased reticulocyte count and osmotic fragility of red cells Jaundice more intense if associated stone in common duct present
9. Hepatitis	Normal or slightly enlarged	Normal or slightly enlarged. May be small in severe case	Slight to intense	Normal to slightly decreased	Normal or decreased	Normal or decreased	
10 Cirrhosis of liver	Slightly to moderately enlarged	Enlarged or small, depending on type and stage of cirrhosis	Absent, slight or moderate depending on type and stage of cirrhosis	Usually slightly, sometimes greatly decreased	Usually decreased	Normal or decreased	Red cells usually normocytic, sometimes macrocytic
11 Congestive splenomegaly	Slightly to greatly enlarged	Normal, enlarged or small	Absent	Usually decreased	Decreased	Decreased	Size of liver and indications of impairment depend on nature of underlying process

FIG 31 14 Chart of important clinical findings in certain diseases showing splenic enlargement.

THE URINARY TRACT. THE ADRENAL GLANDS

THE KIDNEYS

Although serving to focus attention on the kidneys, regional symptoms and local signs, as a rule, are less important in the diagnosis of upper urinary tract disease than the systemic changes created by renal infection and functional impairment, examination of blood and urine, pyelography, and cystoscopy

Pain. Costovertebral and lumbar pain, usually dull and aching, occurs in renal infection such as acute pyelonephritis or abscess, renal distention as in hydronephrosis, perinephric abscess, renal infarct, or large kidney tumor Contrary to popular belief gained chiefly from the "do-you-wake-up-with-a-lame-back?" school of advertising, pain is not a feature of nephritis, although it is occasionally encountered in an acute attack. Lame back is more likely attributable to local musculoligamentous or skeletal disturbance The latter is differentiated from renal pain by associated stiffness, restriction of back mobility, and aggravation by movements necessitating use of regional muscles

Pain originating in the kidney is sometimes felt anteriorly, when it occurs on the right, especially if accompanied by nausea and vomiting, it can be confused with that due to peptic ulcer, gallbladder disease, or appendicitis. Pressure on nearby structures by a large renal mass may produce ill-defined abdominal discomfort and digestive disturbances

Ureteral Colic. Often miscalled renal colic, this is sharp, agonizing, cutting pain originating in the region of one kidney, usually the flank, and extending anteriorly and downward along the course of the ureter, often reaching the homolateral testicle, labia, or thigh It starts and ends abruptly, rarely lasts more than a few hours and, although not definitely rhythmic, tends to show intermittent variations in intensity Between attacks, the patient may be totally asymptomatic or from time to time experience low-grade ache in the loin or lower quadrant. Its downward rather than upward spread and variations in intensity differentiate it from biliary colic; its site, severity, distribution and lack of rhythmic undulation, from intestinal cramps Right-sided ureteral pain is often mistaken for that of acute appendicitis

Disturbance of Micturition. Trouble anywhere in the urinary system may create pain or scalding on voiding, frequency with or without polyuria, oliguria, or anuria. Hesitancy or total failure to void does not necessarily indicate di-

minished urinary secretion; it may be the result of pain, weakness, an obstructive or neurologic lesion affecting the bladder or urethra, diminished mental acuity, or psychologic block. If these can be excluded, complete anuria usually means total renal artery or ureteral obstruction; in acute renal failure, oliguria, not anuria, is the rule. Incontinence may occur in disordered sensorium, neurologic disturbance, and local disease of the bladder or urethra. Often it represents overflow from a distended bladder.

Gross Abnormality of Urine. Discovery by the patient of bloody or cloudy urine may be the initial complaint.

Hematuria. In a high percentage of cases, blood in the urine means serious disease, most likely stone, tumor, tuberculosis, or acute nephritis. It demands prompt, complete investigation. Except when bleeding is profuse, a clue to its source may be provided by the character of the hematuria as indicated below. In the female, catheterization may be necessary to exclude genital tract origin.

1. Anterior urethra. Blood appears chiefly at onset of micturition (*initial hematuria*) or issues from the meatus independently.

2. Posterior urethra. Although there may be some initial hematuria, blood is more concentrated at the end of micturition (*terminal hematuria*).

3. Prostate. Same as posterior urethra.

4. Bladder. Since blood has become mixed with urine, the entire output is discolored but terminal increase is likely.

5. Kidney or ureter. Thorough blending of blood and urine causes uniform discoloration without terminal increase. In acute nephritis, the blood may be changed into acid hematin so that the patient will describe his urine as being "coffee-colored" or "smoky" rather than bloody.

Cloudiness. This may be caused by pus or bacteria, indicating infection somewhere in the tract. Often it is due to precipitated innocuous solutes and serves only as a source of unfounded alarm to the patient.

Hypertension. This is almost always found in acute nephritis, well-established chronic nephritis, chronic pyelonephritis, and other disturbances leading to impaired function. In unilateral kidney disease, especially a small atrophied kidney from some cause such as congenital hypoplasia, chronic unilateral pyelonephritis, or previous trauma, one is likely to find the diastolic pressure relatively high compared to the systolic, and fairly pronounced eyeground changes. When hypertension is discovered, this situation must always be excluded because removal of the diseased kidney may restore blood pressure to normal, and prevent secondary damage to the other kidney.

Tenderness. Costovertebral tenderness of varying degree accompanies renal pain. With ureteral colic, the patient may be tender along the ureter, or in the testicle or labia.

Costovertebral Swelling. Fulness in the costovertebral region is likely in perinephric abscess; fulness or actual bulging, in large renal tumor or abscess.

Spasm. Accompanying costovertebral tenderness, one may find spasm of the regional muscles. Psoas irritation may make the patient unwilling to flex his thigh.

Enlargement of Kidney. The technique of palpating for the right kidney is similar to that used for the liver except that the examining fingers are pressed more deeply into the abdomen and one concentrates on the area lateral to mid-clavicular line. When feeling for the left kidney, it is *preferable to stand or sit at the patient's left,* with one's hands reversed—that is, the right hand in the loin and the left on the abdomen. When the patient takes a deep breath, a palpable kidney, unless it is huge or fixed by inflammatory or neoplastic tissue, will be felt sliding down into the space between the *two hands, it will seem to disappear with expiration.* Usually it is possible to feel only the lower pole but if there is considerable enlargement or unusual mobility, almost all of it can be grasped and the characteristic reniform outline recognized. In the doubtful case, ballottement over the kidney area should also be tried *(see Chap 29).* One might detect an enlarged or displaced kidney by this method, especially if the nonpalpating hand is used to press the costovertebral region anteriorly. Neither kidney is ordinarily palpable unless it is enlarged The one exception to this rule is that in persons who are thin or visceroptotic, especially females, one can almost always feel a normal right kidney, in some instances only during inspiration, in others independently of it The normal left kidney, even in this group, is rarely palpable, although occasionally its tip is detectable on deep inspiration

The usual causes of renal enlargement are infection, especially with abscess formation, malignant tumor, hydronephrosis, and polycystic disease, the last three creating the greatest increase When one kidney is destroyed by disease or removed surgically, physiologic hypertrophy of the other results, but rarely to a degree to make it palpable It is difficult to distinguish between a normal, slightly, and sometimes, moderately enlarged organ One must rely on clinical judgment, with due consideration to the body habitus and presence or absence of factors which might tend to create kidney enlargement Well-established enlargement is appreciable as a mass readily felt in the appropriate region, and perhaps extending downward to occupy a considerable portion of the homolateral side of the abdomen It may be confused with an intra- or retroperitoneal mass or, on the left, with a large spleen. If enlargement is confined to the upper pole, the organ may grow to two or three times its normal size and remain impalpable

X-ray studies are more reliable than palpation There is no technique for exact measurement, one depends on the experience and judgment of the roentgenologist for detection of a slight variant Moderate or great enlargement is easily recognized If the kidney outlines are not clear on a plain film, an intravenous pyelogram may bring them out, dye passing through the kidney substance renders it more radiopaque. If the pelvis is primarily enlarged, this will be apparent on intravenous or retrograde pyelogram In an obscure case, orientation between a large kidney, a large spleen and a mass may be effected by a combination of pyelography, contrast studies of the colon, and perhaps plain films taken after retroperitoneal injection of air. Since the last-named

procedure carries certain hazards such as hemorrhage and air embolism. It should be undertaken only by highly trained personnel

Decreased Size of Kidney. This cannot be detected by physical examination but may be demonstrable by plain x-ray or pyelogram

A single small kidney usually means congenital aplasia, or atrophy representing the end result of previous trauma or some inflammatory process such as unilateral pyelonephritis. Bilaterally small kidneys suggest late chronic nephritis or bilateral pyelonephritis, especially if intravenous pyelography shows impaired secretion of dye.

Manifestations of Impaired Function. These vary with the degree to which excretion of waste products has become limited. As long as the kidneys are still able to meet the ordinary demands of catabolism, no symptoms appear other than, perhaps, nocturia or polyuria But as renal reserve diminishes and toxic substances begin to be retained, the picture becomes one of easy fatigability, headache, vertigo, anorexia, loss of flesh, dryness of skin and mouth, thirst, vague gastro-intestinal disturbances, elevation of blood pressure, and perhaps anemia and edema Acute cases often show oliguria or anuria, chronic cases, polyuria Tests of renal function provide conclusive evidence. When the stage of renal insufficiency is reached and urinary constituents become highly concentrated in the blood, we have the clinical picture known as uremia.

Uremia. Intensification of the above symptoms marks the appearance of uremia. One looks in addition for drowsiness, severe disturbances of vision, intractable headache, hiccough, dyspnea, nausea and vomiting, oliguria, edema, or a hemorrhagic tendency As a rule, any or all of these will appear rapidly in the acute case, gradually in the chronic. In the latter, however, an acute onset may be precipitated by local or general infection, urinary obstruction, or dehydration. Physical signs are restlessness, torpor, dry pallid skin, uriniferous breath, characteristic eye-ground changes (*see* Chap. 4), hypertension, and generalized or dependent edema The most serious case is marked by mental excitement or delirium, coma, muscular twitchings progressing to convulsions, and hemorrhages into mucous membranes and skin In the terminal stage of the chronic case, complicating acute fibrinous pericarditis or acute colitis is likely.

Pronounced elevation of the non-protein nitrogen content of the blood confirms the diagnosis of uremia When this is discovered and no history of previous trouble can be obtained, the hematologic picture is important in differentiating between an acute and a chronic process In the former, hemoglobin and red cells are likely to be normal; in the latter, moderate to profound anemia is the rule.

Edema. This occurs at some stage of all the common types of nephritis. It is most pronounced in acute glomerular nephritis and the nephrotic syndrome Initially collecting where subcutaneous tissues are loose, it is first noticed as periorbital swelling which gives the face a characteristic puffiness Next it involves the ankles, legs, thighs, genitals, and perhaps the trunk In extreme cases, the entire body is swollen, and hydroperitoneum, hydrothorax, and hydropericardium may also be found The skin is tense, dry, cool, and pits on pres-

sure. Edema too slight to alter the patient's outward appearance may be detected as gain in weight. In late chronic nephritis, fluid may first appear in the lungs as a result of left ventricular failure; when the right ventricle has failed, it appears in dependent regions—the ankles and feet of the ambulatory, the genitals, thighs and back of the bedridden patient The face and upper extremities are rarely involved.

Signs of Infection. Septic fever, recurrent chills and leukocytosis are common in pyogenic diseases of the urinary tract. Fever of lesser grade may occur in the acute stages of nephritis and renal tuberculosis.

Changes in Urine. Almost every type of renal disturbance creates some urinary abnormality. albumin, casts, cells, bacteria, or impairment of excretion indicated by fixation of specific gravity, decreased urea clearance, and reduced or delayed phenolsulfonphthalein excretion Albumin, casts, leukocytes, and, less often, a few red cells may appear transiently in the urine of a healthy person, especially following extra physical activity or alcoholic excess, or during any febrile illness. These findings do not necessarily indicate serious trouble Many normal people, especially those who are thin, after remaining upright for a considerable period, will also show albuminuria (*orthostatic albuminuria*); this is not a sign of disease When pus, blood or bacteria are found, catheterization of the bladder, and sometimes of the ureters, is necessary to determine the source. In the female, since they may represent contamination from the vagina, these constituents are significant only if the urine is obtained by catheter, or voided immediately after meticulous cleansing of the vulva

Chemical Changes in Blood. Determination of the non-protein nitrogen and urea levels of the blood is of the utmost importance in the diagnosis of renal insufficiency Other variants which should be looked for are acidosis with reduction of carbon dioxide content, elevated phosphorus with associated diminished calcium, and elevated serum creatinine and uric acid In the nephrotic syndrome, low total protein largely due to diminished albumin, and elevated cholesterol are the rule In calculous disease with radiopaque calculus, determination of calcium and phosphorus levels is essential to exclude disease of the parathyroid glands

Intravenous Pyelography X-ray films of the kidneys and ureters, taken after intravenous injection of a radiopaque dye, make it possible to determine the size of the kidneys, displacement, hydronephrosis, deformity of calices and pelves, width of renal cortices, and presence of ureteral obstruction, irregularity, or dilatation Rapidity and concentration of dye excretion are good indices of renal function, provided the patient has been sufficiently dehydrated to prevent dilution of the material by liberal urinary flow Obviously, if parenchymal impairment prevents concentration of dye, the variants just indicated will be less apparent and perhaps totally undetectable In this event retrograde pyelography is often indicated

Cystoscopy. The use of the cystoscope requires special training; it is not a part of routine physical examination Through the instrument, the urologist inspects the bladder, obtains by ureteral catheterization separate specimens of



FIG 32 1 Intravenous pyelogram showing normal renal calyces and pelves Segments of normal ureters also evident

urine from each kidney, and estimates the secretory powers of each by determining its capacity for dye excretion By introducing a radiopaque solution, x-ray studies of the ureters and kidney pelves and calices are made possible (*retrograde pyelography*) Injection of air instead of a radiopaque substance is sometimes useful in detecting and locating a low-density stone or a small tumor in a ureter or renal pelvis

Panendoscopy. Only through the panendoscope can one obtain a satisfactory view of the urethra This too is a specialized procedure

Congenital Anomalies

Duplication of ureter and kidney pelvis, congenital absence of one kidney, aplasia, fusion of the kidneys across the vertebral column, or, less often, on one side, abnormal rotation, or anomalous location of blood vessels is not un-

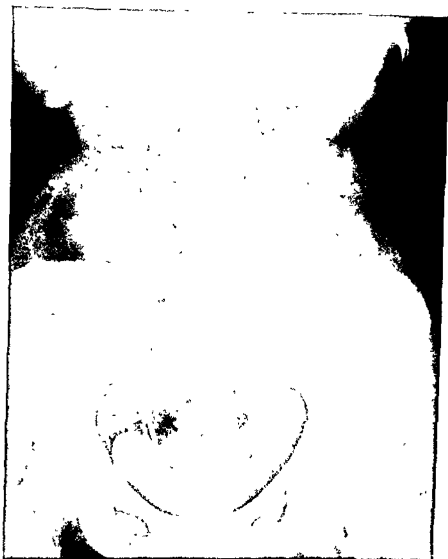


Fig 32 2 Double right kidney and ureter, asymptomatic

common. An anomaly may be asymptomatic, or create trouble by virtue of obstruction, infection, or pressure on an adjacent structure Intravenous and retrograde studies will establish diagnosis

Nephroptosis. In some normal persons and most of those with pronounced visceroptotic habitus the right kidney occupies a low position and is freely movable (*floating kidney*). Easily felt on bimanual palpation, it can be recognized as kidney by its smooth surface, reniform size and shape, and the readiness with which it can be slipped back and forth between the palpating fingers. With the patient supine, it can be manipulated upward toward the normal bed but if he inspires deeply, coughs, or stands up, it will again move downward Too vigorous a squeeze can cause a temporary sickening local abdominal pain.

The degree of mobility can be further evaluated by comparison of intravenous pyelograms taken with the patient supine and upright.

As a rule a low or unusually mobile kidney causes no symptoms although knowledge that he has a "floating kidney" is often a source of concern or vicarious pride to the patient. The drag on its pedicle may create mild lumbar ache, especially when the patient is fatigued or has stood for a long time. Rarely, attacks of acute pain simulating renal colic and accompanied by nausea and vomiting may be initiated by temporary kinking of the ureter with resultant retention of urine in the renal pelvis (*Dietl's crisis*). Changing the patient's position or manipulating the kidney upward may afford relief. In an occasional case, persistently impaired outflow results in some degree of hydronephrosis, with or without infection. Because of the frequency with which the low or fairly mobile kidney is found without symptoms, any surgical interference in a patient whose pain is thought to originate in the kidney should be undertaken only after thorough investigation of his physical and psychologic status, and if impaired drainage can be demonstrated by intravenous pyelogram or retrograde studies

Polycystic Disease. This is familial. Infinite numbers of varying sized cysts develop in the substance of both kidneys. Usually involvement of one is greater than the other Slow, progressive enlargement and parenchymal destruction result from the multiplication and growth of the cysts. Lumbar discomfort, a dragging sensation in the abdomen, noticeable abdominal prominence, symptoms reflecting functional impairment, or an episode of gross hematuria may be the first hint of trouble. On examination, moderate or great enlargement of one or both kidneys will be found; either may be so huge that it fills the homolateral side of the abdomen Its surface will be definitely irregular Readily detected bilateral enlargement, especially if the kidney surfaces are irregular, is all but pathognomonic of cystic disease Periodic gross or microscopic hematuria occurs in about one-third of the cases; eventually urine and blood studies will show evidence of parenchymal insufficiency. In the later stages secondary infection is likely Pyelograms disclose enlargement of the kidneys with elongation of the major and irregular dilatation of the minor calices.

NEPHRITIS

This term refers to bilateral, diffuse, non-pyogenic kidney disease. The diversity of etiologic factors, variability of the clinical picture, complexity of structural changes in the kidney, and frequent difficulty of correlating clinical and pathologic findings have made it impossible to devise any simple grouping which will include every case. For routine clinical purposes, the following classification is satisfactory:

1 Glomerular nephritis.
2 Nephrotic syndrome.
3 Acute renal shut-down
4 Vascular nephritis.

Glomerular Nephritis. Also called *hemorrhagic nephritis*, this results from



Fig 32 3 Polycystic disease of kidneys Intravenous pyelogram Dilatation, elongation and deformity of calyceal systems Borders of enlarged kidneys, evident on original film, indicated by dotted lines

some acute infection, almost always beta-hemolytic streptococcus: sore throat, acute tonsillitis, scarlet fever, or a skin infection It may appear in an acute form, or be discovered by urinalysis and other studies during or shortly after such an episode Or it becomes manifest in a less acute form at a later date without, because of its mildness, having been detected at onset In this event, correlation between the nephritis and previous acute infection may be impossible to establish, but it is nonetheless generally agreed that such a relationship always exists.

Acute Stage. Although onset is occasionally unattended by symptoms and the disease is discovered on routine examination of the urine, the usual early phase is marked by appearance of bloody, "smoky," or brownish urine, diminished excretion, and edema first noticed as puffiness beneath the eyes but soon becoming generalized Headache, malaise, loss of appetite, and fever are the rule. Nausea and vomiting, bilateral aching lumbar pain, and urgency and frequency of urination may accur Puffy, non- or slightly pitting edema, and rising blood pressure are the outstanding signs The urine, in addition to blood cells,

contains albumin, leukocytes, hyaline and bloody casts, and, later, granular and epithelial casts. In its severe form, acute glomerular nephritis may be regarded as a systemic disease affecting the vascular system generally. Most cases recover completely, usually after a period of months. In a small proportion, death from uremia, congestive heart failure, or, rarely, cerebral hemorrhage will occur. Others develop a progressive disease, the features of which are described herewith.

Chronic Latent Stage. In some instances, the patient has presumably recovered from an acute attack and feels well. In others there is no history of previous trouble; the disease is discovered incidentally. The only evidence of the process is persistence in the urine of albumin and usually small numbers of red cells and casts. Renal function is not impaired. Occasionally, after three or more years complete healing may occur. More likely, however, the urinary findings persist for a long time; the patient remains symptom-free but blood pressure may begin to rise. Persons in this group are subject, especially following respiratory infection, to periodic exacerbations of the disease, or attacks in which they show the characteristic features of the nephrotic syndrome (*see below*) Eventually the terminal stage is reached unless death occurs earlier from cardiac failure secondary to hypertension, cerebral vascular accident, intercurrent infection, or some unrelated trouble

Terminal Stage. This can occur during the acute phase, after 2–3 years of low grade acute nephritis, or after a longer period during which the patient has been in the chronic latent phase. The process is the same; the time interval reflects the degree of activity of the renal disease Some symptom of renal insufficiency, such as headache, fatigue, weakness, impairment of vision, anorexia, hiccough, nausea, nosebleed, vertigo, polyuria, or nocturia may be the first hint of trouble. Often the seriousness of the situation is brought out by studies of renal status prompted by accidental discovery of some variant such as elevated blood pressure, persistent albuminuria, eye ground changes or anemia The well-advanced case shows hypertension with secondary cardiac hypertrophy, retinopathy, pallor, perhaps puffiness of the face and dryness of skin, and some degree of anemia Characteristic urinary changes are low specific gravity, small amounts of albumin, and variable numbers of red cells, leukocytes and casts. The course is irregular and marked by frequent attacks of upper respiratory or other incidental infection which appear to accelerate the nephritic process, or initiate nephrotic episodes Gradual reduction of renal function ultimately causes death, although, as in the latent stage, cardiac failure, cerebral vascular accident, or some unrelated trouble may supervene before the kidneys reach the stage of total inadequacy.

Nephrotic Syndrome. As already indicated, a certain number of patients with glomerular nephritis may, especially following respiratory infection, develop episodes of this variant It is also encountered in diabetes (*Kimmelstiel-Wilson disease*), amyloidosis, multiple myeloma, disseminated lupus erythematosus, toxemia of pregnancy, and rarely in syphilis and certain less common diseases. Very rarely, the syndrome has been reported to follow an immunization

procedure, and prolonged administration of trimethadione. The *characteristic features are massive edema, marked albuminuria, low serum protein largely due to loss of albumin, and a high serum cholesterol* The predominant symptoms are the discomfort of the edema, and general weakness. Examination shows generalized, usually pronounced edema and a characteristic waxy pallor which is out of proportion to the degree of anemia. Blood pressure may or may not be elevated Urine is scant and contains a large amount of albumin, variable numbers of casts—hyaline, granular and fatty—doubly refractile lipoids, abundant white and epithelial cells, but a few or no red cells. Blood non-protein nitrogen is normal. Anemia is not striking Basal metabolic and blood protein-bound iodine readings are low; here they do not represent diminished thyroid function. Radioactive iodine uptake is normal or occasionally slightly elevated.

When it appears in glomerular nephritis this phase often passes after a few months, but may recur from time to time Meanwhile, the patient continues in a state of latent or active chronic nephritis which ultimately reaches the terminal phase.

Except in glomerular nephritis, the nephrotic syndrome is to be regarded not as a primary renal disturbance, but as secondary to disease elsewhere Search must be made for and treatment directed at the underlying trouble If the latter is cured, the renal changes disappear; if not, one usually finds downward progression with subsequent death from renal failure, intercurrent infection, or other process related to the basic disease

Diabetic Nephropathy (Kimmelstiel-Wilson Disease) This has come to the front within the last decade or two as a common cause of the nephrotic syndrome Perhaps its appearance is related to the longer life span of the diabetic, incident to insulin thrapy Although usually encountered in long-standing diabetes, it is occasionally seen in a case of short duration, but here it might be postulated that the diabetes itself had gone unrecognized for a long time Like retinopathy, neuropathy, and arterial disease, it is regarded as a degenerative complication; the retinal and renal changes are especially likely to coexist. In contrast to nephrosis due to most other causes, hypertension and indications of impaired renal function are the rule *Necrotizing papillitis (see below)* is sometimes a terminal complication.

Acute Renal Shut-Down. Under this term we include disturbances also known as *lower nephron nephrosis, acute renal insufficiency*, and *acute tubular failure* Common causes are incompatible transfusion, sulfonamid sensitivity, poisoning by a heavy metal or other chemical, especially carbon tetrachloride or a glycol, toxemia of pregnancy, and any disturbance, especially severe trauma, which creates low blood pressure or peripheral circulatory failure. The underlying process is probably a direct toxic effect on tubular epithelium, or renal ischemia. The first manifestation is usually abrupt diminution of urinary output to 25–200 cc. daily, complete anuria is rare Headache, nausea and vomiting, tremors or muscular twitching, cramps, insomnia or diminished sensorium, and other indications of uremia are the rule. At the start, blood pressure is likely to be normal or low but may gradually increase to a moderate

hypertensive level. Edema is not a feature unless parenteral fluid is too generously administered. What urine is obtained is cloudy because of large amounts of debris, and shows specific gravity fixed at a low level, moderate albumin, red cells, white cells, tubular epithelial cells and, of great significance, heme-pigment casts. The blood non-protein nitrogen rises rapidly and to a high level; serum potassium may become elevated. The patient may die within a few days of renal failure alone, or renal failure plus other toxic effects of the etiologic agent. In the former, hyperkalemia is regarded as an important factor. Others, after a period of 1–4 weeks but averaging about 10 days, will begin to show increased urinary output, a highly favorable sign. General symptoms and evidence of the uremic state begin to disappear, but only after diuresis has been maintained for several days. During the early period of diuresis, uncontrolled loss of electrolytes may result in serious depletion and collapse. If the patient survives the oliguric and early diuretic stages, gradual recovery is the rule. Specific renal function tests may continue to show some residual damage but the patient's health is usually unaffected.

A picture closely resembling this disturbance occurs in epidemic hemorrhagic fever beginning usually about the fourth day (see Chap. 3).

Vascular Nephritis. Most cases in this group represent the end-result of changes associated with hypertension (*renal arteriolosclerosis*), or, less often, generalized arteriosclerosis. As a rule, parenchymal damage is not enough to create symptoms referable to the kidneys. Their involvement is detected by studies of urine and renal function. The possibility of progressive parenchymal disease and eventual renal insufficiency is always in the background. Although polyuria or nocturia secondary to impaired concentration may be an early symptom it is more likely that one related to vascular trouble elsewhere—headache, diminished visual acuity, angina pectoris, nocturnal dyspnea—will first bring the patient to his physician. Except in malignant hypertension, death, in contrast to hemorrhagic nephritis, is more likely to result from congestive or coronary heart disease, or cerebral vascular accident, than from renal failure.

Urinary findings are a small amount of albumin and a few hyaline or granular casts and leukocytes. Specific gravity may or may not be low. Red cells are usually present in appreciable numbers, but not grossly. One exception is that gross hematuria may appear in malignant hypertension, presumably from rupture of a vessel within the kidney.

A clinical pattern comparable to that just described is occasionally encountered in polyarteritis nodosa, gout, lupus erythematosus, and, due to renal calcinosis, in hypercalcemia due to hyperparathyroidism, vitamin D intoxication, or other cause.

Renal Congestion

Myocardial insufficiency or some other process interfering with return blood

of red cells Dye excretion may be diminished or delayed. The concentration test is usually normal, although in severe congestion specific gravity may not reach maximum levels.

Renal Infarction

Multiple infarcts of the kidneys are not infrequent in cases of chronic heart disease, especially when auricular fibrillation is present, and in subacute bacterial endocarditis. These are usually relatively small, often asymptomatic, and rarely cause appreciable functional impairment Sometimes the patient will complain of sudden sharp costovertebral pain which subsides within a few hours but leaves a dull ache for 1–2 days Local tenderness may be elicited. Transient hematuria, usually microscopic, occasionally gross, occurs in a high percentage of cases; when found under appropriate circumstances it is to be regarded as diagnostic.

A large infarct secondary to impaired blood flow through a major vessel, most likely from large embolus or dissecting aneurysm, causes severe local pain and tenderness, and perhaps evidence of peripheral circulatory failure The affected kidney, due to seriously disturbed physiology, will show little or no dye excretion on intravenous pyelography, but a retrograde pyelogram will be normal Death in uremia may occur from sympathetic shut-down of the opposite kidney. If the patient recovers and his kidney is sufficiently scarred, subsequent pyelographic studies may show deformity and dilatation of one or more calices, with perhaps regional irregularity of the cortical margin

A comparable picture cay be caused by thrombosis of a renal vein secondary to an inflammatory lesion of the kidney or extension upward of thrombophlebitis from a leg or the pelvis

Renal Infection

Acute Pyelonephritis. This is a diffuse infectious process involving the pelvis and parenchyma of one or both kidneys It is more common in females Since the trouble is not confined to the renal pelvis, the term "pyelitis" often applied to it should be discarded The disease may begin spontaneously or be preceded by infection elsewhere, especially in the respiratory, gastro-intestinal, or lower urinary tract, the prostate or seminal vesicles. In such instances, it is usually bilateral. It can also result from impaired renal drainage, here whether it is uni- or bilateral depends on the site of the obstructive lesion It is a frequent complication of pregnancy, presumably due to partial ureteral block by pressure of the enlarged uterus Sometimes it follows urinary tract instrumentation The usual infecting agent is the colon bacillus, less frequent are streptococcus, staphylococcus, *Bacillus proteus*, and *Bacillus pyocyaneus* Infection may be mixed.

Onset is abrupt and attended by fever, recurrent chills, often dull lumbar pain, and leukocytosis This disease is one of the commonest causes of repeated chills Micturition is frequent and painful Except for moderate, sometimes exquisite tenderness in the costovertebral angle, no physical signs are found.

The urine contains pus, bacteria, and, depending on the severity of the infection, a few or many red cells. More than one examination may be necessary; if temporary block occurs on the diseased side, a single specimen obtained may be exclusively from the non-diseased kidney and hence be normal. The severity and duration of the course vary. Recovery time, formerly averaging 2–3 weeks, has been appreciably shortened since the introduction of sulfonamides and antibiotics. Recurrences are common, particularly in the presence of any factor favoring poor drainage. Blood stream infection may be a serious *complication*, especially when the organism is sulfonamide- and antibiotic-resistant. The greatest danger is the development of chronic pyelonephritis. For this reason, complete investigation, to exclude an obstructive lesion or a focus of infection, must always be carried out in the recurrent or therapy-resistant case.

Necrotizing Papillitis. A fulminating form of acute pyelonephritis encountered in diabetics, usually but not necessarily with Kimmelstiel-Wilson disease, this is marked by suppuration and necrosis of the renal pyramids, in addition to the usual changes of acute pyelonephritis. As a rule, the picture is one of a severe intractable systemic infection superimposed on that of ordinary

Resistance to spe-

provement may be

biotic, relapse will

follow its withdrawal. As a rule, pyelographic studies are not indicated, if, for some reason, they are performed, irregularity of calices secondary to destruction of papillae will be seen. Death from overwhelming infection or renal failure is the end-result. Occasionally, the disease follows a less acute form with exacerbations and remissions, but is ultimately fatal.

Chronic Pyelonephritis. Low grade infection with slow but progressive destruction of renal parenchyma may persist for years. It may begin insidiously or develop after an acute attack, especially one which is inadequately treated or in which impaired drainage is a factor. For a matter of years, the patient may feel in good health or somewhat below par, but have no specific complaints. Exacerbations of acute pyelonephritis are likely, especially following intercurrent respiratory or enteric infection, undue fatigue, or other factor imposing stress on his machine. Functional impairment secondary to parenchymal destruction may eventually lead to death in uremia. Or serious hypertension, developing presumably from associated renal vascular changes, will cause fatal termination from one of its other complications prior to onset of kidney failure. Calculus formation is possible. The late stage may be indistinguishable from that of advanced glomerular nephritis. Except during an acute exacerbation when pus and bacteria appear, the urine shows nothing characteristic. Albuminuria, a few leukocytes, perhaps red cells, and a few hyaline and granular casts may be found, but cultures are usually negative. Complete investigation of the urinary tract is always indicated, especially to exclude an obstructive lesion which might be contributing to the persistence of the process and its failure to respond to specific therapy. Intravenous or retrograde pyelography will show uni- or bilaterally small kidney with cortical narrowing, and blunting,

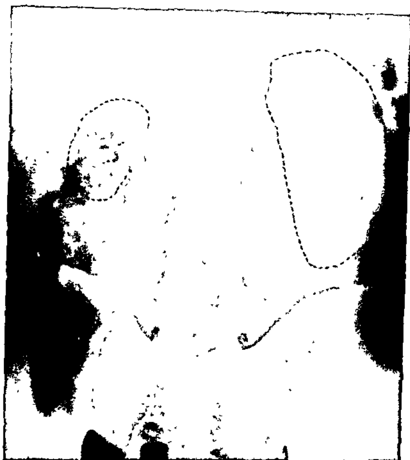


Fig 32 4 Chronic unilateral pyelonephritis in man age 60 with hypertension for one year Retrograde pyelogram, right kidney (Intravenous pyelogram a few days before this film was taken showed normal excretion by hypertrophied left kidney, no excretion on right) Renal borders, evident on original film, indicated by dotted lines Left kidney Compensatory hypertrophy Right kidney Parenchyma decreased in thickness as indicated by short distance between calyces and outer edge Calices somewhat blunted, infundibuli crowded together Blood pressure returned to normal shortly after right nephrectomy and has remained so for 7 years Anatomic diagnosis Healed pyelonephritis, secondarily contracted kidney

loss of cupping, and dilatation of calices If obstruction is a factor, dilatation of the pelvis will be evident.

Unilateral pyelonephritis is one of the causes of a small contracted kidney with which one may find hypertension, usually with diastolic pressure relatively high compared to the systolic, fairly pronounced eye ground changes, and albumin and casts in the urine Surgical removal often results in pronounced lowering of pressure and prevents progressive disease in the remaining kidney

Abscess of Kidney. Multiple Abscesses Multiple abscesses of one or both kidneys are common in septicemia, especially when due to staphylococcus They may also follow a remote local pyogenic lesion such as a furuncle, carbuncle, or infected finger; here the primary focus is sometimes insignificant and apt

to precede the renal trouble by weeks or longer. The picture is that of sepsis alone, or sepsis plus flank pain, tenderness, and spasm. Urinary findings are not striking; a few leukocytes and red cells are likely. Staphylococcus or other pyogenic organism may be recovered by urinary culture. *Perirenal infection* from rupture of an abscess through the capsule is a possible complication.

MASSIVE ABSCESS Massive abscess (*septic infarct, renal carbuncle*), a large discrete suppurative mass occupying one-half to one-third of the kidney substance, *represents enlargement of a single or confluence of several small abscesses*, occurring secondarily to a remote focus as described above. Here, too, symptoms are likely to appear weeks after subsidence of the primary infection. The usual picture is one of moderate to severe lumbar pain, costovertebral tenderness, perhaps spasm of regional muscles, and systemic indications of sepsis Palpable enlargement of the kidney and visible swelling in the loin are possible. Urinary findings are rarely helpful, sometimes staphylococcus or other pyogenic organism is recoverable by culture. Plain films may show an enlarged or irregular kidney outline and obliteration of the psoas muscle shadow; pyelograms, destruction of calices in the affected segment.

Perinephric Abscess. As in abscess of the kidney, the perinephric tissues are most likely to become infected from a remote focus. Less often, rupture of a cortical abscess or pyonephrosis through the renal capsule is the cause Because *fever and other systemic symptoms often precede local indications for* days or weeks, and pain, if present, is poorly localized, the diagnosis is apt to remain obscure for some time. Eventually pain, costovertebral tenderness, and perhaps fulness in the loin crystallize the picture Psoas muscle irritation may create pain on extension of the thigh. Except for a few leukocytes, the urine is negative and its cultures sterile. As a rule, plain x-ray shows faint kidney outline and psoas shadow, and local curving of the spine with its convexity away from the affected side. Impaired motion of diaphragm is sometimes demonstrable. Pyelography is rarely helpful Negative x-rays do not exclude the disease.

Tuberculosis. Renal tuberculosis is secondary to active or inactive tuberculous infection elsewhere, usually of lung, bone, or lymphnodes. It is becoming less frequent in this country because of better control measures, and earlier recognition and treatment of other forms of the disease

Since tuberculosis of the kidneys is regarded as a blood-borne infection, it is thought that cortical lesions may, in the presence of tuberculosis elsewhere, be much more common than is generally supposed. This premise is based on the fact that tubercle bacilli can sometimes be discovered in the urine by direct examination, culture or guinea pig inoculation, in patients with disease elsewhere but with no other indication of urinary tract involvement. It is further thought that such cortical lesions may heal without causing permanent renal damage; the tuberculous bacilluria will disappear. This is borne out by the frequency with which healed lesions are found *post mortem* in patients dying of pulmonary or other active form of tuberculosis In other cases, the process will progress in one or both kidneys and lead to the formation of large areas of

necrosis, caseation and scarring, often with calcification. At times a kidney may be completely destroyed and become a sac of necrotic material comparable to pyonephrosis.

Until the renal disease is well-established, there are no symptoms or signs other than perhaps mild fever, fatigue, and other indications of low-grade infection, but these are more likely a reflection of some other focus of activity. Bacteriologic study of the urine prompted by knowledge that a focus does exist in a lung or elsewhere may lead to the detection of early renal disease. Otherwise attention is first called to the kidney by the appearance of frequency and dysuria, discovery of albumin or pus in the urine, an attack of hematuria, or, less often, pain in the kidney region The dysuria and frequency usually represent involvement of the bladder, although some authorities believe that they may be due to bladder irritability caused by urinary toxins originating higher up. Relief is not obtained by therapeutic measures ordinarily effective in cystitis. Frequency and dysuria not attributable to pyogenic infection or an obstructive lesion, usually mean tuberculosis At this stage, pus is almost always present in the urine; red cells are possible. One or more episodes of gross hematuria may occur In well-advanced cases, one finds systemic signs of serious infection and, in bilateral involvement, renal insufficiency. The kidney is tender and sometimes palpable Compensatory hypertrophy may cause some enlargement of the less involved organ

A purulent urine which proves sterile on culture for the ordinary pyogenic organisms demands exclusion of renal tuberculosis

X-ray studies are not helpful until the calices and pelvis are involved, so that negative findings do not exclude early tuberculosis On pyelography the first appreciable change is blunting and loss of cupping of a calix with evidence of destruction of the papilla Later the calix becomes dilated and the infundibulum leading to it, narrowed and irregular With progression, more calices become similarly involved, and gross alterations in renal architecture are evident In advanced disease, parenchymal calcification and scarring may lead to a shrunken non-functioning organ Involvement of ureter and bladder can cause obstruction which, in turn, creates a large kidney similar to that seen in pyonephrosis, but usually with some degree of calcification in addition

The only conclusive evidence of tuberculosis is demonstration of tubercle bacilli in the urine by direct smear, culture, or guinea pig inoculation.

Obstructive Renal Disease

Hydronephrosis. Interference with free flow of urine creates dilatation of pelvis, calices and tubules, and secondary parenchymal atrophy The greatest dilatation occurs in long-standing partial ureteral obstruction Even when block is complete, total parenchymal atrophy does not occur for a long time so that urinary secretion continues and the kidney eventually becomes a thin-walled sac As a rule, because of the impaired drainage, some degree of secondary infection is superimposed and contributes to the destructive process The common causes of hydronephrosis are:

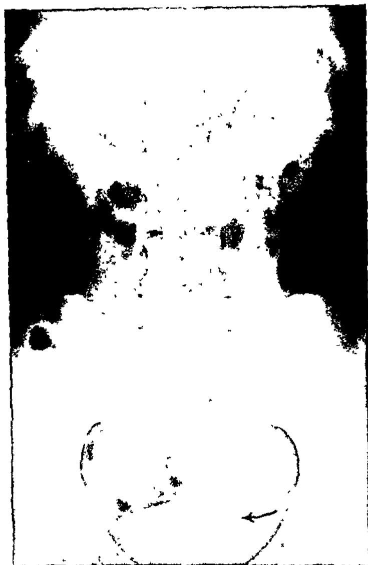


FIG 32 5 Left hydronephrosis, slight, due to partial ureteral obstruction by radiopaque stone (arrow) Intravenous pyelogram Pelvis and calices somewhat dilated Ureter shows increased caliber above stone, it is not visible below Very little dye remaining in right kidney since its drainage was not delayed

1. Prolonged impaired bladder emptying, as from obstructing prostate, urethral stricture, or some neurogenic disturbance The bladder distends to accommodate retained urine but symptoms such as dysuria, frequency, or difficult micturition invite attention to the trouble which, with the exception of some neurogenic disorders, can probably be corrected. For this reason, hydronephrosis rarely develops to the degree which pertains in the causes listed below and



FIG 32 6 Left hydronephrosis, pronounced, due to obstruction at ureteropelvic junction (arrow) by aberrant renal vessel Retrograde pyelogram Pelvis and calyces show marked dilatation and loss of normal patterns

is of less importance than the changes created by renal infection secondary to the stasis (pyelonephritis)

2. Obstruction within a ureter or kidney pelvis by stone or, rarely, primary neoplasm.

3 Some congenital variant such as narrowing of a ureteropelvic junction, ureterocele, congenital dilatation of a ureter, or aberrant renal artery causing compression of the upper ureter.

4. External pressure on a ureter or renal pelvis by a pregnant uterus, tumor, or inflammatory mass, or invasion by malignant tumor of a pelvic or abdominal structure

5. Stricture of a ureter due to cicatricial contraction caused by intrinsic inflammation as in impacted stone or tuberculosis, or by spread of infection from a diseased appendix, sigmoid diverticulum, or other nearby structure

6. Ureteral kinking in nephroptosis This is rare

Acute obstruction, by causing local pain, tenderness, and perhaps nausea and vomiting, is usually discovered early, and is likely to be corrected before hydronephrosis becomes a major problem.

In slowly developing hydronephrosis based on partial obstruction, symptoms may be absent or the patient subject to attacks of mild to severe loin or anterior abdominal pain and tenderness, perhaps with nausea and vomiting. Especially if the pain is anterior, cholecystitis, peptic ulcer, or appendicitis is apt to be suspected. Attacks are prone to be initiated by undue exertion; rarely, they are relieved by change of position which so alters the relative positions of the structures involved as to reduce the obstruction temporarily. Relief associated with temporary output of a large quantity of urine is said to be a common sign but actually this is rare. Whether or not the kidney is palpable obviously depends on the extent to which it has enlarged; discovery by the patient of fulness in his abdomen is occasionally the first sign of trouble. Hematuria may occur, especially following trauma. If the hydronephrosis is infected, attacks simulating acute pyelonephritis are likely. The urine may be normal, show a few red cells, or evidence of infection. Functional impairment depends on the duration and degree of obstruction and infection. In unilateral disease, the healthy kidney will carry most of the excretory load so that the extent of damage to the diseased kidney can be detected only by pyelography and split function studies. Plain x-ray films will show a large kidney, intravenous pyelography, narrowing of cortex, dilated calices and pelvis, and dilatation of ureter above the point of obstruction. In the advanced case, poor concentration of intravenously administered dye makes retrograde studies necessary.

Pyonephrosis. This can be regarded as an end-result of renal infection, usually of the obstructive type, or renal tuberculosis. Thick pus fills the dilated pelvis and destruction of kidney substance is relentless, eventually the organ becomes a sac of pus with no remaining parenchymal tissue. Indications of chronic sepsis and, in bilateral disease, functional impairment, are evident. Costovertebral pain and tenderness are the rule but both are likely to be absent when a kidney is completely destroyed. The diseased organ may or may not be palpable. Pus and bacteria predominate in the urine. By cystoscopy one will find pus emanating from the ureteral orifice. The x-ray picture is indistinguishable from that of hydronephrosis.

Calculus Disease

One or more stones form in the pelvis or calices of a kidney and remain there. But at any time a small one may descend into the ureter and either be retained, or sooner or later continue downward into the bladder where it can remain or be expelled through the urethra. In most cases, some systemic or local disturbance can be found to explain the formation of stones. Since their chemical constituents vary from case to case, chemical analysis of any stone which is passed or removed surgically is always indicated, as it will provide a definite clue to the nature of the underlying disease. The following classification of calculi is used by Dr. Fuller Albright and his associates in the Stone Clinic at the Massachusetts General Hospital.

Calcium Phosphate, Calcium Oxalate, or a Combination of Them. These stones are the result of hypercalcinuria which, in turn, in most likely due to one of the following:

1. Prolonged immobilization.
2. Hyperparathyroidism.
3. High milk or cheese intake.
4. Vitamin D intoxication.
5. Sarcoidosis.
6. Idiopathic hypercalcinuria, a syndrome of unknown etiology characterized by urine calcium excretion of more than 150 mg daily, normal blood calcium, low blood phosphorus, and associated *Staphylococcus albus* urinary tract infection.
7. Renal tubular acidosis, a rare disease of unknown cause perhaps related to previous pyelonephritis, and marked by indications of tubular insufficiency without glomerular insufficiency. The striking features may be repeated attacks of generalized weakness or paralysis based on low blood potassium, osteomalacia or (in children) rickets, or symptoms reflecting renal calculus. Chemical studies of the blood show low carbon dioxide with high chloride, low pH, low potassium, normal calcium and low phosphorus. Urinary findings are poor concentration, neutral or only slightly acid reaction in spite of blood acidosis, and high calcium output.

Pure Calcium Oxalate. This stone is usually single, recurrences are not common. It is usually found in a patient who becomes repeatedly dehydrated, such as a vigorous athlete, truck driver, or stationary fireman. In contrast to the cases noted above, urinary calcium excretion and blood electrolytes are normal. Whether or not high oxalate intake is a factor is unsettled.

Mixed Magnesium, Ammonium, and Calcium Phosphate. This type is associated with chronic urinary tract infection by urea-splitting organisms, especially *Bacillus proteus*, *Bacillus pyocyaneus*, and *Escherichia coli*, as in a patient requiring repeated catheterization, in-dwelling catheter, or prolonged kidney drainage. The so-called *stag-horn* stone, occupying the renal pelvis and extending into the calices, is confined to this chemical group. Some observers believe that a stone composed of these constituents is often superimposed on a previously existing calculus caused by hypercalcinuria, cystinuria or uric acid and, in such instances, represents a complication secondary to infection.

Cystine. The relatively rare cystine stone is presumably the result of an obscure, probably heredo-congenital renal disturbance which hampers normal resorption of cystine and certain other amino acids. Cystine, which is not a normal urinary constituent, can always be found in the urine. These stones are often multiple and likely to begin in childhood. In the family of a patient with a cystine stone, some members may have stone, others cystinuria without stone, and still others, no evidence of the trait.

Uric Acid. This type, usually multiple, is found only in a persistently acid urine. Although uric acid stones have been regarded as closely associated with gout, it now seems well established that actually they are encountered in only a small number of patients with this disease. Furthermore, when they are present, hyperuricemia or clinical gout is unlikely. Uric acid stones sometimes occur in ulcerative colitis and regional enteritis.

When calculus disease is encountered, every effort should be made to establish the underlying factor Chemical analysis of a spontaneously passed or surgically removed stone has already been mentioned. The patient should be carefully questioned regarding prior prolonged immobilization, excessive ingestion of milk, cheese, or any alkaline remedy, recurrent attacks of local sepsis, such as prostatitis, skin infection or infected teeth, symptoms hinting at gout, and any illness in his family suggestive of stone or gout When hypercalcemia is suspected, one should look especially for band keratopathy (*see* Chap. 4), and bone tenderness which may be a precursor of spontaneous fracture secondary *to calcium loss* *Studies of the urine should include examination for oxalate,* triple phosphate, calcium phosphate, cystine and uric acid crystals, cultures, repeated pH determinations for the persistent alkalinity of urea splitting infection or the persistent acidity associated with uric acid stone, and the chemical test for cystine In the presence of hypercalcinuria, determinations of daily urinary calcium excretion, and calcium, phosphorus, and phosphatase blood levels are indicated. The usual values associated with the disturbances noted are shown in the accompanying chart. When calcium excretion is high, bone x-rays, especially of skull, spine, and teeth (for status of laminae durae), should be taken

Clinical Manifestations

Renal or ureteral stone often exists for years without causing symptoms Sometimes it is detected during investigation prompted by incidental discovery of a urinary abnormality Other cases are heralded by pain, gross hematuria, or frequency or urgency resulting from reflex bladder irritation. A small stone lodged in a ureter or ureteropelvic junction causes attacks of ureteral colic, with perhaps intervening dull lumbar or abdominal aching Distension and

DISTURBANCE	SERUM CALCIUM	SERUM PHOSPHORUS	SERUM AKALINE PHOSPHATASE	24-HOUR URINE CALCIUM EXCRETION
Immobilization	Normal or increased	Normal or increased	Normal	Greatly increased
Excessive milk intake	Normal	Normal	Normal	Increased
Hyperparathyroidism	Increased	Decreased	Normal or increased	Increased
Sarcoidosis	Increased	Decreased, normal or increased	Increased or normal	Greatly increased
Vitamin D intoxication	Increased	Increased	Normal	Greatly increased
Idiopathic hypercalcinuria	Normal	Decreased	Normal	Increased
Burnett's syndrome	Increased	Normal	Normal	Decreased or normal
Renal tubular acidosis	Normal	Decreased	Normal or increased	Increased

FIG 32 7 Important chemical findings in disturbed calcium metabolism (Prepared with assistance of Dr Philip H Henneman)

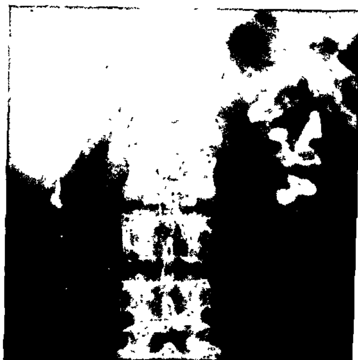


FIG 32 8 Radiopaque stag-horn calculus, left kidney, in a patient with hyperparathyroidism Several isolated stones are visible in right kidney region Loss of calcium from vertebrae also evident

obstipation associated with acute pain may create a picture confusable with acute appendicitis, intestinal obstruction, or other intra-abdominal insult With a large stone in the kidney pelvis, dull loin pain is the rule—colic unlikely Tenderness is possible in the costovertebral region or, with ureteral colic, along the course of the ureter or in the homolateral testicle or labia In the long-standing case, hydronephrosis with palpable kidney, secondary infection, and functional impairment are possible If both ureters become completely obstructed or one is obstructed and the function of the opposite kidney seriously impaired, anuria will occur Rarely, this is the first symptom Depending on the status of the case, the urine may be normal or show blood, pus, or evidence of parenchymal damage

In almost every instance the presence of urinary calculus can be confirmed by x-ray A radiopaque stone will be evident on plain films but pyelographic studies are advisable to determine the exact site of the shadow with respect to the urinary tract, and thus to exclude phlebolith, calcified lymphnode or other innocuous cause A non-opaque stone can be detected by conventional or, in the doubtful case, air pyelogram

Related Disturbances

A discussion of calculus disease cannot be regarded as complete without mention of the following related renal disturbances:

Nephrocalcinosis. In the presence of hypercalcinuria or hypercalcemia, crys-

talline calcium deposits in the kidney substance can lead to gradual impairment of renal function and, unless the disturbance is discovered early and promptly treated, terminate in renal insufficiency. Stones as such may or may not be present On a plain x-ray film, one may see multiple minute flecks of density with fan like distribution representing calcium deposits in the renal pyramids; when seen end-on, they have a circular arrangement In the advanced case diffuse density throughout the medulla is apparent.

Uric Acid Deposits. In long-standing gout, deposition of uric acid crystals in the interstitial tissues of the kidneys is not uncommon It creates a progressive inflammatory process which eventually leads to scarring, atrophy, and severe functional impairment.

Sulfonamide Deposits. A decided hazard in the use of sulfonamide drugs is the accumulation of free and acetylated crystals in the calices and pelves A sludge is formed which, since the process is usually bilateral, leads to rapid development of oliguria or anuria If recognized early, it can often be relieved by prompt irrigation of the renal pelves, otherwise death from renal failure is highly probable During sulfonamide therapy this complication can usually be averted if the urine is kept alkaline, and free flow maintained by high fluid intake

Burnett's Syndrome. This is thought to occur primarily in patients with some degree of underlying renal impairment, most likely old pyelonephritis It is characterized by hypercalcemia but, in contrast to most other disturbances with elevated blood calcium, urinary calcium excretion is not increased, and blood phosphorus not diminished Other features are alkalosis, usually band keratopathy, and rapidly developing uremia

Tumor of Kidney

Adenoma. Small multiple adenomas of the kidney are not infrequently discovered *post mortem* Occasionally one will grow sufficiently to create a palpable mass, perhaps with dull pain, gross or microscopic hematuria may occur. Exploration is usually required to distinguish it from a malignant lesion

Hemangioma. Usually a small tumor located near a renal papilla, this is an occasional cause of unexplained hematuria Diagnosis is rarely made without operation

Solitary Cyst. Although not a tumor in the strict sense, cyst is included here for convenience It is usually asymptomatic Its importance stems from the fact that it is often accidentally discovered by pyelography and can be distinguished from malignant tumor only by exploration, or perhaps aortography In the latter, cyst may show a localized area of diminished vascularity, malignant tumor, one of irregular increased vascularity

Embryoma (Wilms' Tumor). Usually occurring in infancy or early childhood, this is a mixed tumor with the sarcomatous element predominating It is extremely malignant, rapidly growing, and frequently fatal, although in a few cases diagnosed early, proper therapy may effect a cure As a rule, the tumor is not discovered until it has grown large enough to create abdominal heaviness,



FIG 32 9 Large renal cell carcinoma, left kidney Retrograde pyelogram Lateral displacement of caliceal system with compression, narrowing, and elongation of pelvis (lower arrows) and spreading of upper calices (upper arrows) Border of enlarged kidney, evident on original film, indicated by dotted line

pain, or palpable mass X ray studies will show a grossly enlarged kidney with distorted calyceal pattern

Renal Cell Carcinoma. Formerly called *hypernephroma,* this is often well advanced before being discovered Intermittent hematuria with, as a rule, long intervals between episodes is usually the initial symptom Since the blood arises in the kidney, the urine is uniformly discolored Except for possible colic due to passage of a clot, pain is a late feature A dull ache in the loin or anteriorly will reflect pressure on adjacent structures or drag on the renal pedicle, sharp lumbar pain radiating anteriorly will reflect nerve trunk invasion or pressure In a small percentage of cases, a palpable kidney is the first sign Often local manifestations of trouble are preceded by some systemic sign of malignant disease such as weight loss, anemia, or weakness, or, because of the frequency with which the renal vein is invaded by tumor, evidence of metastatic growth in a lung, bone, or elsewhere Consequently, the diagnosis of renal cell carcinoma is often made by search for the primary focus prompted by discovery of a metastatic lesion Sometimes the tumor extends through the renal vein into the

inferior vena cava; a deep lateral abdominal mass associated with signs of inferior vena caval obstruction is always suggestive of renal cell carcinoma If the tumor is in the left kidney, varicosities in the left side of the scrotum or the left broad ligament may be evident as a result of impaired blood flow through the spermatic or ovarian vein X-rays will show enlargement of the kidney, irregularity of its outline, and compression, distortion, and perhaps filling defects of calices and pelvis.

Sarcoma of renal parenchyma or invasion by lymphoma can create a picture resembling renal cell carcinoma As a rule, the diagnosis can be established only by operation, or biopsy of a growth elsewhere

Tumor of Renal Pelvis. This appears in a papillary or non-papillary form. The latter is extremely malignant and metastasizes rapidly by lymphatics or blood stream The papillary tumor is also malignant but less so than the non-papillary, it often shows a tendency to reseed down the ureter and sometimes into the bladder Hematuria, often intermittent, is the outstanding symptom. Local pain, if present, is usually a reflection of ureteral obstruction Diagnosis is established by demonstration of a filling defect of the renal pelvis on conventional or air pyelograms

BLADDER

Suprapubic, perineal, or genital pain, disordered micturition, or a change in appearance of the urine may call attention to the bladder but does not necessarily mean that it is the primary seat of the trouble Any of them may be a reflection of vesical disturbance secondary to renal, prostatic or spinal cord disease, intrapelvic tumor, or some intrapelvic inflammatory process such as salpingitis, tubo-ovarian abscess, diverticulitis, colitis or pelvic appendicitis Rectal and vaginal examinations, x-ray studies, cystoscopy, sigmoidoscopy and sometimes lumbar puncture may be necessary for diagnosis

Frequency of Micturition. This may be associated with increased or scanty output *Temporary polyuria* is common in high-strung persons during periods of emotional stress It also occurs following excessive fluid intake or administration of a diuretic, during convalescence from certain febrile illnesses, when edema is disappearing and, in some women, during or just after menstrual period. It is occasionally encountered in intermittent hydronephrosis and following an attack of migraine or epilepsy *Constant polyuria* is found in certain types of nephritis, uncontrolled diabetes mellitus, and diabetes insipidus *Frequent scanty urination* is common in healthy persons who are nervous or lie awake at night In disease, it is a symptom of vesical or urethral inflammation, external pressure on the bladder, or retention with overflow

Painful Micturition. Usually associated with frequency and difficulty in starting the stream, this is most likely to be encountered in bladder or urethral irritation from any local cause such as cystitis, trigonitis, tuberculosis, calculus, ulcer, tumor, urethritis, or caruncle The discomfort is often maximal at the end of urination and may, especially if the bladder neck is acutely inflamed, be attended by a series of exquisitely painful spasms (*strangury*)

Incontinence. Unconscious operation of its reflex can cause a normally full bladder to empty itself. This is common in children, especially at night, and is also encountered in stuporous or comatose states, and in certain diseases of the central nervous system A bad fright may cause involuntary urination in a normal person In women, as a result of relaxed or injured urinary sphincter due to previous childbirth, escape of a small amount of urine following cough, sneeze or laughter is common. Postmicturitional dribbling in males is evidence of relaxed external sphincter usually due to a nerve disorder or lack of muscle tone, at times it is associated with chronic prostatitis or prostatic enlargement True incontinence must be distinguished from involuntary or voluntary passing of small amounts of urine due to overflow, or frequent bladder contraction and impaired sphincter control from acute inflammation

Pain. Acute inflammation or acute distention of the bladder creates pain in the suprapubic region, perineum, genitals and, sometimes, the rectum.

Retention. Unless retention is complete or causes vesical enlargement sufficient to be recognized on abdominal examination (*see below*) it can be diagnosed only by catheterization If a catheter is introduced after the patient has attempted to empty his bladder, retained urine will be withdrawn (*residual urine*) Complete retention must not be confused with anuria due to renal insufficiency or ureteral block, here the bladder contains no urine, or only a scant amount

ACUTE This is usually complete The patient feels an urge to urinate but cannot do so Regional pain is often severe A full bladder is often the cause of restlessness in a stuporous or semicomatose patient. The common causes are

1. Functional disturbance of bladder reflex following operation or labor, and in stuporous or comatose states
2. Paralysis of bladder mechanism in acute disease of the spinal cord such as acute poliomyelitis, and following spinal cord injury
3. Prostatic abscess
4. Vesical or urethral stone
5. Complete shutdown of partial retention
6. Traumatic rupture of bladder or urethra, with extravasation of urine into surrounding tissues.

CHRONIC. Usually partial, this develops gradually. It is indicated first by diminution in force or size of the stream, later by frequent voluntary or involuntary urination Pain is not common because the bladder, having time to stretch, develops greater capacity Parenchymal renal damage resulting from impaired outflow, secondary infection, or both, is a likely complication If chronic retention becomes complete, the picture changes to that of acute retention The common causes are

1. Benign hyperplasia of prostate
2. Urethral stricture
3. Chronic spinal cord disease, such as tabes dorsalis.
4. Diverticulum of bladder.

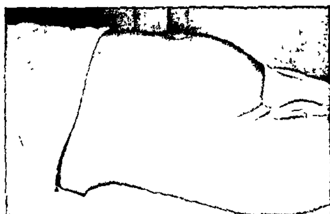


FIG 32 10 Rounded prominence of lower abdomen due to distended bladder

5. Cystocele In contrast to the causes noted above, this variant, although a nuisance to the patient, does not have serious complications

In a doubtful case, differentiation between obstruction and impaired nerve control as the cause of retention can be accomplished by cystometric studies In the former, the pressure will rise as the bladder is filled; in the latter, no appreciable change will be evident

Distention. A distended bladder is recognized by palpation as a smooth, rounded, firm, symmetrical tumescence extending upward above the pubes It may be barely perceptible or, in the extreme case, extend to the umbilicus or even higher Unless the abdominal wall is unusually thick, it is often visible as a rounded prominence The percussion note is dull; when enlargement is not sufficient to produce a palpable mass, it can sometimes be diagnosed by finding a circumscribed area of slight dulness above the symphysis. These findings may all be obscured by ascites, intestinal distention or obesity. When found in a man, they almost always indicate a distended bladder, in a woman, the possibility of a pregnant uterus, or pelvic cyst or tumor must also be entertained In the doubtful case, catheterization is essential.

Because ascites can conceal a distended bladder, abdominal paracentesis must never be performed until the bladder has been emptied by the patient's own effort or, when retention is suspected, by catheterization

CYSTITIS

Since the normal vesicular mucous membrane is resistant to infection, bacterial cystitis is rarely a primary disease. One must look for some underlying cause including

1. Recent repeated or in-dwelling catheterization
2. Incomplete emptying, as in prostatic obstruction or cystocele.
3. Urethritis.

4 Intravesical disease such as foreign body, diverticulum, tuberculosis, or tumor.

5. Disease of upper urinary tract, especially pyelonephritis.

6 Infectious or neoplastic disease of a genital organ.

7. Extension into bladder of a gastro-intestinal lesion such as diverticulitis or carcinoma.

8. Some systemic disease, particularly diabetes mellitus.

Complete urologic and general investigation is always indicated in any case of cystitis which does not promptly respond to therapy, or becomes recurrent

Acute Cystitis. The outstanding symptom is frequent, painful micturition, the pain being particularly pronounced toward the end of the act In a severe case, persistent bladder spasm may cause, without relation to micturition, constant, intense pain referred to the perineum or genitals, and perhaps suprapubic ache or pai

tenderness i

of frank blood may be forced out at the end of urination. At cystoscopy, the mucosa shows diffuse reddening and small ulcerations which bleed easily

C

of frequent and urgent passage of small amounts of urine, perhaps accompanied by pain. The only signs are those found by cystoscopy—chronic inflammatory changes of bladder mucosa with, sometimes, small cystic buds (*cystitis cystica*) or deposits of crystalline material (*encrusted cystitis*) on its surface In long-standing cases where obstruction has not been prominent, a contracted bladder of small capacity may be created by chronic fibrosis The urine contains leukocytes, rarely, gross pus, and bacteria. If a purulent urine is sterile on culture for ordinary pyogenic organisms, tuberculosis and, in the male, chronic prostatitis should be suspected.

Interstitial Cystitis. A disease of unknown etiology, this is almost entirely confined to women, it must be excluded in every case of severe intractable cystitis. The symptoms are persistent frequency and urgency, often with severe pain aggravated by micturition The urine usually contains a few leukocytes but is not grossly purulent, hematuria is possible Organisms may or may not be recovered on culture On cystoscopy one finds a small bladder with a thin mucosa showing small shallow ulcerations surrounded by hyperemia, and areas of scarring representing healed ulcers

Tuberculosis

Tuberculosis of the bladder is always secondary to disease elsewhere, usually the kidneys, sometimes a genital organ It is marked by frequent, painful micturition and other symptoms of cystitis but, in contrast to other forms of bacterial cystitis, is resistant to the conventional therapeutic agents. It must be excluded in the presence of pyuria which is sterile on culture for pyogenic organisms, or resistant to treatment Tuberculosis may be strongly suspected if,

by cystoscopy, ulcerations or lesions resembling tubercles are seen, but the diagnosis must be confirmed by recovery of tubercle bacilli from the urine.

Vesical Calculus

Stone in the bladder, single or multiple, is usually evidence of urinary stasis or infection It is most commonly associated with prostatic or urethral obstruction but is also encountered in diverticulum and atony Sometimes a stone descended from the kidney will remain in the bladder and grow. An occasional cause is deposition of urinary salts around a foreign body or on an ulcerated lesion due to tuberculosis, other infectious process, tumor, or radiation. Bladder stone is relatively rare in women, probably because obstruction is less common The usual symptoms are painful and urgent urination which varies with the severity of secondary infection, pain on jolting, and perhaps intermittent sudden cessation of the urinary stream with or without resumption of flow on change of position Symptoms are less pronounced when the patient is recumbent or inactive The urine contains pus, and often blood. Whether or not a stone can be seen by x-ray depends on its size and density. Diagnosis can be established by cystoscopy

Diverticulum

Diverticulum begins as a small outpocketing of the bladder wall, gradually increases, and may even become larger than the bladder itself Since it appears to be related to obstruction at the bladder outlet, it is largely confined to men in the older age group Diverticulum may exist for years without causing symptoms but infection secondary to retention eventually sets in, and the picture becomes that of cystitis. Pyuria is the rule, hematuria occasional. Secondary stone or bladder tumor is a not infrequent complication. Occasionally a diverticulum grows in such a way as partially to obstruct a ureter, causing dilatation and infection of the homolateral kidney The diagnosis is made by cystoscopy and cystography.

Cystocele

Downward bulging of the bladder floor results from weakening of its supporting structures, usually by childbirth The patient complains of frequency, incontinence under such stress as cough or laughter, and "something protruding" in the vagina On vaginal examination one will find downward bulging of the anterior vaginal wall which will become more pronounced when the patient strains. Catheterization usually reveals slight retention; secondary infection eventually occurs Cystogram may show abnormally low bladder neck

Vesical Tumor

To all intents and purposes, tumor of the bladder is almost always epithelial and to be regarded as malignant It may be papillomatous or infiltrative. Tumor is usually well-established before signs of trouble appear The classic initial sign is an episode of hematuria which usually begins suddenly, is fairly profuse,

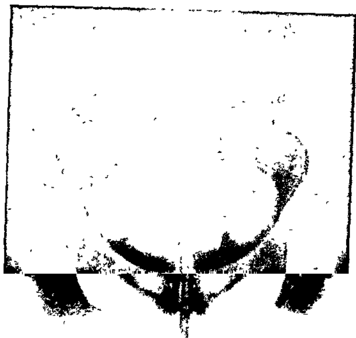


FIG 32 11 Diverticula extending from right and left sides of bladder Radiopaque medium introduced through catheter

and suddenly ceases Here we have one of the important reasons why blood in the urine always demands prompt and thorough investigation. Recurrences become increasingly frequent, between them, microscopic hematuria may persist Occasionally urinary irritability or infection is the first manifestation Physical examination is rarely helpful, although in the advanced case, an area of induration representing tumor in the bladder wall or invaded perivesical tissues may be palpable by rectum or vagina Contrast x-rays may show irregularity of bladder wall, or a filling defect Diagnosis is confirmed by cystoscopy Metastasis to regional nodes is common, to remote organs, occasional Direct extention to perivesical structures and bones of the pelvis also occurs Hydronephrosis secondary to invasion of a ureteral orifice is another likely complication

Perforation or Rupture

Perforation may occur as the result of a puncture wound from without, or, rarely, from sequestrum in osteomyelitis of a pelvic bone. It can also happen in malignant disease. Rupture is possible by a sudden blow or great pressure on the abdomen but only if the bladder is full Tear of the vesical floor or urethra may complicate fracture of the pelvis

The patient usually cannot void, or passes small amounts of bloody urine A specimen obtained by catheter is scant and bloody. Profound shock is the rule.



Fig. 32.12 Carcinoma of bladder. Film taken 30 minutes after intravenous pyelogram shows large irregular filling defect of floor and left side.

If the perforation is above the peritoneal line, urine extravasates into the peritoneal cavity, giving a picture suggesting general peritonitis; if below the line, extravasation into the tissues above the symphysis pubis or in the perineum causes redness, edema, and perhaps tumor-like swelling. Diagnosis is confirmed by air-cystography which will show air in the perivesical or neighboring retro- or intraperitoneal spaces.

URETHRA

The more common urethral disturbances are discussed in connection with the male and female genital organs (*see* Chap. 33).

ADRENAL GLANDS

Acute Cortical Insufficiency (Adrenal Crisis)

Adrenal crisis may be superimposed on chronic or latent cortical insufficiency, or occur independently. In the former it is induced by stress with its increased demand for cortical hormone, it can follow surgical operation, trauma, or hemorrhage, but may also be precipitated by some minor incident such as a head cold, gastro intestinal upset, or dental extraction. In the absence of underlying insufficiency, an acute crisis can be caused by surgical removal

sult of acute parenchymatous degeneration, apart from hemorrhage, in any overwhelming infection, including meningococcemia.

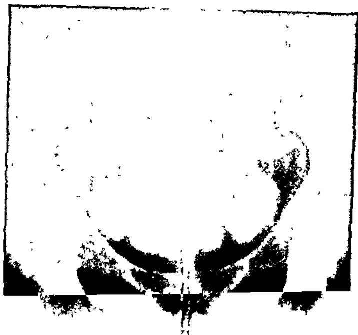


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Profound weakness, semi-coma, headache, perhaps costovertebral angle pain, nausea, vomiting, and diarrhea appear suddenly. Physical examination shows hyperpyrexia, hypotension, *dehydration, and* rapid, often feeble heart action. The systolic pressure is usually 80 mm or lower, the diastolic 10–0 mm. During the first few hours of an acute crisis, the diagnosis must be established on the clinical picture alone. Blood findings may not be help[illegible]

[illegible] potassium and non-protein nitrogen, absence of marked reduction in the number of circulating eosinophiles

Rapid decline with circulatory collapse, perhaps subnormal temperature, coma, and death were formerly the rule Currently, thanks to adrenalcortical hormones, antibiotics, vasopressor drugs, and blood and electrolyte replacement therapy, disaster can often be averted by prompt treatment.

The current widespread therapeutic use of ACTH and cortisosteroids makes it essential to emphasize that adrenal crisis can be easily created by too rapid withdrawal, especially following long-term administration Either during or after withdrawal, operation, trauma, infection or other added stress even of a minor nature greatly *increases the hazard* A crisis can usually be averted by resumption or increase of the hormone dosage to meet the anticipated requirement

Chronic Primary Cortical Insufficiency (Addison's Disease)

About half the cases are due to cortical atrophy of unknown cause Most of *the remainder result* from parenchymal destruction by tuberculosis, here one can usually obtain a history of tuberculosis or exposure to it, or evidence of the disease elsewhere in the body, most likely a kidney Rarely, the glands are replaced by neoplasm, especially metastatic malignant tumor, or destroyed by amyloid disease, scleroderma or hemochromatosis.

Onset is usually insidious with darkening of skin, easy fatigability, and increasing weakness appearing first. Later come loss of weight, anorexia, bouts of nausea, vomiting, diarrhea and abdominal pain, nervousness, irritability, and mental fatigability or difficulty of concentration Symptoms are worse in very hot or cold weather. A careful history may disclose episodes based on hypoglycemia such as hunger, headache, visual disturbance, greater weakness, sweating, trembling, emotional upset, apprehension or even unconsciousness. These are apt to occur in the late morning, late afternoon or following extra exertion The weakness and fatigability predominate later in the day, in contrast to psychogenic disturbances in which, as a rule, they are most pronounced in the morning Muscular cramps secondary to sodium chloride deficiency and reduced blood flow are possible. Costovertebral pain and tenderness may be present. Sometimes the first indications of trouble are those of an acute crisis developing during a period of extra though often minor stress imposed by one of the causes noted in the preceding section.

Characteristic physical signs are pigmentation, hypotension, evidence of weight loss, muscular weakness and, usually, dehydration. Pigmentation may be diffuse or spotty and is typically of a sun tan or chestnut-brown hue; occasionally it is yellowish, as in pernicious anemia. Favored sites are the buccal mucosa, knuckles, knees and elbows, recent scars, folds of the skin, and areas where clothes press tightly. The areolae, nipples and perianal, perineal and genital areas where the skin is normally dark may become almost black. Diagnosis in the absence of pigmentation in the mouth is doubtful. The skin is fine and shiny. Cranial hair becomes darkened and graying delayed. Especially in women, body hair is diminished, the forearms may be devoid of the normal growth, axillary and pubic hair is scant and fails to grow after shaving. In some cases, the cartilage of the ears is hard and inflexible due to calcification or perhaps actual ossification. The heart may appear small to percussion. Systolic pressure is usually below 90 mm., diastolic, below 70 mm., and pulse pressure also diminished. These variants may be evident with the patient recumbent but if not, will appear when he rises to a sitting or standing position and remain until he lies down again. In contrast, the normal or arteriosclerotic patient may show a temporary pressure fall when he first stands up but it will last for only a few moments.

The basal metabolic rate is usually 5–20 per cent below normal. The blood chemical changes noted above are found in the advanced case but are not necessarily striking in the earlier phases. By x-ray the heart shadow may be small and calcification is sometimes observed in the adrenals, the latter is not diagnostic since it can occur in other disturbances. Diagnosis can be confirmed by the various procedures designed for evaluation of adrenal cortical function such as assay of urine 17-hydroxycorticosteroids and 17-ketosteroids, assay of blood 17-hydroxycorticosteroids, and the Thorn 48-hour ACTH, Robinson-Kepler-Power water, and Albright insulin tolerance tests.

In the past, without the currently available therapeutic agents, the course was downward, likely to be marked by acute crises, and eventually fatal. Now, happily, good health can be maintained for years by appropriate replacement treatment.

With involvement of the adrenals by metastatic malignant disease, amyloidosis and the other disturbances mentioned above, the typical picture of Addison's disease does not develop but functional impairment of the glands undoubtedly contributes to the weakness, manition, and other late systemic manifestations.

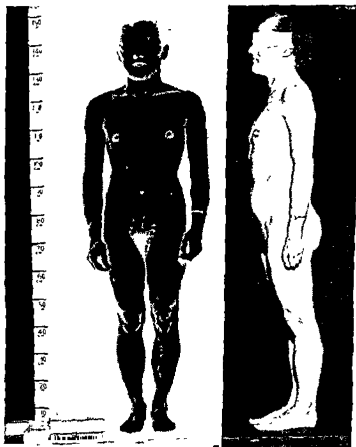
Secondary Cortical Insufficiency

In anterior pituitary insufficiency any combination of trophic gland deficiency may be encountered. In such disturbances as Simmonds' disease, Sheehan's syndrome, and panhypopituitarism one may find evidence of adrenal impairment along with that of hypofunction of other glands, especially the thyroid and gonads. As a rule, the gonads are the first to fail and the adrenals the

last In contrast to primary cortical insufficiency, pigmentation and electrolyte changes are less striking; carbohydrate and basal metabolic, and gonadal changes, more prominent.

Cortical Hyperfunction

Cushing's Disease or Syndrome. Excess secretion of adrenal cortical hormones may be created by basophilic adenoma of the pituitary (*Cushing's disease*), or primary hyperplasia or tumor of the adrenal cortex (*Cushing's syndrome*) The picture is of obesity, plethora, weakness, hirsutism, striae, thinning of skin, easy bruising, hypertension, accelerated vascular aging, osteoporosis, amenorrhea or impotence, and obvious or latent diabetes (*see* Chap 3). The blood changes include elevated carbon dioxide, low potassium, erythrocytosis, and polymorphonuclear leukocytosis with diminished lymphocytes and eosinophiles. The glucose tolerance test shows a hyperglycemic response, the insulin tolerance test, insulin resistance Pyelographic or perirenal pneumographic studies may show evidence of tumor



[illegible] drome due to adrenocortical hyperplasia Female, age 17 Short [illegible]

[illegible] in reproduction. (Courtesy Dr. Janet W McArthur)

Adrenogenital Syndrome. Bilateral adrenal hyperplasia with excessive secretion of adrenal androgen may occur as a congenital abnormality. In girls it is marked at birth by enlarged clitoris, and some anatomic variant of the urogenital sinus such as a single perineal orifice into which both the vagina and urethra open (*pseudohermaphroditism*) Other features are precocious growth and general development, early appearance of axillary and pubic hair, and signs of masculinization such as hirsutism and deep voice. In the boy, general development, growth of penis, and appearance of secondary sex features are precocious Rarely, especially in males, there may be an accompanying diminished secretion of the salt factor leading to symptoms of Addison's disease, and perhaps death in an acute crisis Most of the changes indicated are reversible and can be controlled by long-term corticosteroid therapy.

In adults adrenal virilism is most likely due to primary adrenal carcinoma.

Primary Carcinoma. The clinical picture is usually a mixed one presenting some of the meatures of virilism, and some of Cushing's syndrome In the female, typical changes are deepening of voice, masculine growth of hair on face, trunk, and extremities, perhaps with temporal and vortical balding, atrophy of breasts, increased muscularity, amenorrhea, and enlargement of clitoris Such features of Cushing's syndrome as obesity, facial acne, hypertension, mild polycythemia, diminished glucose tolerance, and perhaps glycosuria may be found Although in the young male, precocious general and primary and secondary sexual development is apparent, in the adult adrenal virilism is apt to go unnoticed for some time so that primary carcinoma is more likely to be overlooked It is to be suspected if a man appears to have good nutritional status and muscular development in association with symptoms suggesting advanced malignant disease. Appearance of gynecomastia may provide a clue. Metastases to regional nodes and lungs are the rule.

Diagnosis can sometimes be established by the various tests of adrenal function, especially if they are supported by x-ray evidence of kidney displacement or by pneumographic demonstration of a mass Surgical exploration may be necessary

Metastatic Carcinoma. In primary carcinoma, especially of a lung or breast, metastatic invasion of the adrenals is a not infrequent *post-mortem* finding As noted above, the picture of adrenal insufficiency is not likely, although impairment of the glands may contribute to the late symptoms

Disease of Adrenal Medulla

Discussion of the adrenal medulla can be limited to three types of tumor Of these only pheochromocytoma produces symptoms reflecting hormonal imbalance There is no known disorder which creates hypofunction.

Ganglioneuroma. A benign tumor usually found in childhood, this gives no symptoms unless it becomes large enough to create a palpable mass, evidence of pressure on a nearby structure, or x-ray indication of displaced kidney.

Sympathetoblastoma. This is a highly malignant tumor also seen chiefly in the young A palpable mass or symptoms of regional pressure may be the initial hint of trouble. Sometimes systemic manifestations of malignant disease, such

as weakness, anemia, or loss of weight, or evidence of metastatic growth in the skull, liver, eye, lung, or long bone appear first.

Pheochromocytoma. The manifestations are a *reflection of excess secretion* of epinephrin and nor-epinephrin Although, as a rule, the tumor arises in the adrenal medulla, it sometimes occurs elsewhere, the most common aberrant site being just above the bifurcation of the aorta Occasionally two tumors are found, one in each adrenal or in some other combination of sites Typically the patient *has recurrent attacks of hypertension, often extreme, with intervals* of normal pressure between, or transient episodes of pronounced pressure elevation superimposed on an underlying hypertensive state. In some cases, episodes are not striking; the hypertension is indistinguishable from the usual benign or malignant form Actual attacks are marked, in addition to blood pressure rise, by other indications of hyperadrenalism such as severe headache, dizziness, dyspnea, palpitation, tachycardia, tremulousness, and perhaps paresthesias, nausea, vomiting or diarrhea Anxiety is striking, in fact, the patient is often incorrectly thought to be suffering from an anxiety neurosis Features not ordinarily found in hypertension which may invite attention to the possibility of pheochromocytoma, especially if they become accentuated during a hypertensive crisis, are. excessive sweating, intermittent attacks of peripheral vasoconstriction and vasodilatation, *fall of systolic and diastolic pressures with rise of pulse rate* when the patient changes from supine to upright, elevated basal metabolic rate, irregularly elevated fasting blood sugar level sometimes sufficient to cause glycosuria, and perhaps a diabetic glucose tolerance curve In the patient subject to them, episodes may be precipitated by physical strain, lying in a certain position, pressure or massage *over the tumor area, or emotional upset In the* untreated case, death can occur from any of the complications of severe hypertension such as cerebral vascular accident or cardiac failure

The diagnosis can usually be established by careful evaluation of the clinical picture. X-ray is rarely of assistance, although a large tumor may create kidney displacement or be detected by *retroperitoneal pneumogram Help may* be obtained by special procedures such as the histamine test which will precipitate an attack, and the benzodioxane test which will create a sharp drop in blood pressure. These are not without danger and should be performed only by an experienced person under controlled conditions In recent years, assays of epinephrine and norepinephrine content *of the urine have largely superseded* these tests In the pertinent case, detection of elevated excretion of either or both in a 24-hour specimen, collected during or between episodes, virtually confirms diagnosis of pheochromocytoma

THE ANUS, RECTUM, AND GENITAL ORGANS

ANUS AND RECTUM

Tragic diagnostic errors often arise from failure to examine the rectum There is indeed wisdom in the dictum credited to various medical sages of the past to the effect that the first duty of the consultant is to perform a rectal examination. The latter includes

1 Inspection of mucocutaneous region
2. Digital palpation
3 Inspection of anal canal with the anoscope
4 Inspection of rectum and lower sigmoid with the sigmoidoscope

Inspection of the orifice and, unless there is danger of stirring up trouble, digital palpation should always be performed in a routine physical examination. Whether instrumentation should be employed depends on the nature of the problem Introduction of one's finger or an instrument must be carried out with extreme care, sometimes it should be postponed as, for example, in acute fulminating colitis where instrumentation might provoke hemorrhage, or in angina pectoris where an attack of pain might be precipitated by stretching a tight anal ring Depending on circumstances, one or more of these procedures is always necessary in the following situations.

Irritation of Mucocutaneous Region. Itching or burning may be due entirely to a local disturbance, or be a reflection of trouble higher in the intestinal tract, some systemic disease, or a generalized cutaneous disorder

Pain. Painful defecation, pain in the region of the rectum, and tenesmus arise from a variety of causes, the most common of which are fissure or fistula in ano, hemorrhoids, ischiorectal abscess, carcinoma of rectum, impacted feces, and diarrhea from any cause Disease elsewhere in the pelvis, such as cystitis or prostatitis can also refer pain to the rectal region.

Change in Bowel Habits (*see* Chap 30)

Melena (see Chap 30)

Local Discharge or Swelling. Discharge, swelling, mass, or tenderness in or around the anus can usually be explained by some local trouble such as ischiorectal abscess, fistula, hemorrhoids, colitis, or carcinoma

Suspected Disease of a Regional Structure. In such diverse troubles as acute

pelvic appendicitis, prostatic infection or hypertrophy, tumor of bladder, disease of a female genital organ, and peritoneal carcinomatosis, key information can often be obtained by rectal investigation.

TECHNIQUE OF EXAMINATION

Inspection of Mucocutaneous Region

The Sims' position is preferable. The patient lies on his side with the dependent leg slightly flexed at hip and knee and the other flexed to approximately 15 degrees so that from the knee down it rests on the table, not on the dependent extremity. The upper part of the trunk is slightly rotated, bringing the chest nearer the table. The buttocks are drawn apart and the area inspected for

1. Blood or purulent discharge.
2. Pallor, thickening and excoriation of mucosa and perianal skin characteristic of pruritus
3. Pinworms or other parasites
4. Condylomas (*see* Chap. 3).
5. Edema, swelling, or redness on either side of the orifice suggestive of perianal or ischiorectal abscess
6. External hemorrhoids, protruding internal hemorrhoids, or tabs representing the end-stage of thrombosed hemorrhoids
7. External opening of a fistula, usually a minute hole
8. Break in the mucous membrane (*fissure in ano*) or local mucosal thickening which might be concealing a fissure This is most likely found at the anterior or posterior commissure but may be elsewhere
9. Prolapsed rectal mucosa

Fissure, prolapsing internal hemorrhoids, or prolapsing mucosa may be visible only if one stretches apart the borders of the anal ring, sometimes downward straining by the patient is also required

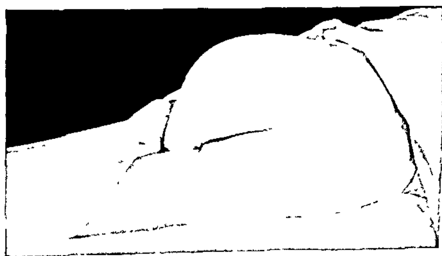


FIG 33 1. Sims' position

Digital Palpation

When one is concerned chiefly with the rectum itself, or a structure high in the pelvis, the patient should be in the Sims' position, or on his back with legs and thighs flexed. For examination of the prostate and seminal vesicles, urologists prefer to have him standing with trunk bent forward, or in the knee-chest posture; these positions are less desirable when the object of search is a lesion high up because the rectum and intrapelvic structures tend to fall away from the examining finger Little can be accomplished unless the patient is relaxed and the finger insertable without causing pain

The anal ring and gloved finger should be generously lubricated with a suitable surgical jelly The patient is then instructed to strain gently, and as he does so the finger is gradually and with care inserted through the canal One must be sure that the lubricant is worked into the latter, not scraped off at its entrance. Unless the sphincter is spastic, the ring narrowed by scar tissue, or some painful lesion such as thrombosed hemorrhoid or fissure interferes, the finger will pass through the anal canal without appreciable discomfort If severe pain is provoked, the attempt should be abandoned To be surveyed are

Anal Sphincter. A *tight* sphincter is most often due to prolonged diarrhea, or failure for years to have formed movements because of injudicious use of cathartics It is also caused by fissure in ano or other inflammatory lesion and is frequently encountered in nervous, sensitive persons without local disease Spasm may be sufficient to prevent insertion even of the little finger A *relaxed* sphincter can be found in association with a relaxed perineum due to childbirth, or with internal hemorrhoids or prolapsed mucosa Pronounced loss of tone suggests some neurologic lesion

Anal Canal. An inelastic fibrosed ring with narrowed lumen is frequently encountered at the external orifice It represents contracted scar tissue usually from previous inflammatory process, or trauma, especially tear during labor. Internal hemorrhoids, usually not palpable, can occasionally be felt as thickened folds, especially if the finger is swept in circular fashion around the inside of the canal Induration about a chronic fissure may be felt usually at one of the commissures, rarely, it is possible for the trained finger to recognize local thickening about the internal opening of a fistula Inward bulging and tenderness of the wall suggest ischiorectal or perianal abscess

Rectal Wall and Lumen. The finger should pass over the entire surface, first with the patient relaxed, then while he gently strains down Straining may force within reach a tumor otherwise impalpable Variants which might be discovered are

1 Impacted fecal mass, or foreign body

2 Stenosis due to annular carcinoma or some chronic inflammatory lesion such as ulcerative colitis, syphilis, tuberculosis, or lymphogranuloma venereum.

3 Rectal polyp, felt as a soft, pedunculated, freely movable mass.

4 Carcinoma, presenting as a firm, nodular, elevated growth often partially obstructing the lumen Occasionally, it is spongy and cauliflower-like.

5. Distention or ballooning of the rectum, said to be a sign of peritoneal inflammation.

Prostate Gland and Seminal Vesicles. Size, consistency, shape, and tenderness are noted The seminal vesicles are rarely felt unless diseased (*See* below.)

Uterus and Adnexa. As in vaginal palpation, examination of these parts is performed bimanually (*see* below). Position, size, shape, consistency, tenderness, and mobility are noted Care must be taken not to confuse the cervix, which is readily felt, with a tumor of the anterior rectal wall Such variants as retroversion, tumor of ovary, or inflammation of tubes and ovaries are sometimes better appreciated by rectum than by vagina.

Pelvic Cavity. Tenderness high on the right may establish a diagnosis of pelvic appendicitis when, in the suspected case, tenderness is not found on abdominal palpation. In appendiceal abscess, downward bulging of the pelvic floor or a fluctuant mass may be discovered A similar picture on the left can be created by diverticulitis of the sigmoid

Metastatic malignant disease of the pelvic peritoneum may be felt through the rectal wall—usually the anterior—as one or more hard nodules which sometimes partially encircle it to form a palpable ridge (*rectal shelf*) In the female, softer, less firmly fixed nodules behind the cervix usually mean endometriosis Widening of the cervix and thickening and nodular infiltration of the broad ligament, detectable only by rectum, may be the first local confirmatory sign of cancer of the endocervix In the male, hard, nodular infiltration felt above the prostate is likely to represent extension of bladder carcinoma.

Anoscopy

Introduction of the anoscope or sigmoidoscope should be preceded by digital examination, since besides contributing information, this step somewhat dispels the patient's apprehension, dilates the sphincter, and helps grease the canal Either the knee-chest or Sims' position is suitable Direct or reflected light can be used The instrument should be warmed, thoroughly lubricated, and inserted slowly Each quadrant of the wall is investigated in turn; it is more comfortable for the patient if, instead of rotating the anoscope in the canal, it is removed and reinserted for each quadrant One looks particularly for.

1. Fissure in ano which might have escaped previous inspection and palpation.
2. Anal polyp—a small, fibrous, pedunculated growth
3. Internal hemorrhoids—purplish-red, linear swellings where engorged veins lie under the mucosa These run lengthwise along the canal and are likely to be grouped.

Sigmoidoscopy

A cleansing enema should precede this examination except in the presence of an acute inflammatory disturbance The knee-chest position is preferable The patient kneels on the table with his thighs vertical, his head turned to one side and resting on the table, and the upper part of the chest as close to the



Fig 33 2 Correct position of patient for sigmoidoscopy

table as possible. In this position, the rectum and sigmoid tend to straighten out, and, later, when the obturator is removed, air will flow in through the instrument and somewhat distend the bowel. Here too, the instrument should be warmed and well lubricated. Some clinics prefer a special table so designed that the patient kneels on a horizontal platform, with his trunk bent downward at a 45 degree angle

The patient is asked to strain downward, and as he does so, the sigmoidoscope is gently and slowly inserted Once the sphincter is passed, straining is no longer necessary The instrument is carefully advanced for a few centimeters until stopped by the sacrum, whereupon the obturator is removed With its base slightly elevated so as to direct its tip downward, it is then carefully maneuvered, under direct vision, along the curves of the bowel until it has been inserted to full length Difficulty is often encountered at the promontory of the sacrum, or elsewhere But, unless one meets obstruction or stenosis, or the patient is extremely tense, complete insertion can usually be accomplished with the aid of careful manipulation and perhaps inflation of bowel with air *Undue pressure should never be used nor large amounts of air injected*, either may cause rupture of bowel or some other catastrophe Obviously, no attempt should be made to force the instrument beyond an obstructive lesion. One notes the distance to which the tube was passed and looks especially for the following:

1 Cause of failure to pass the sigmoidoscope to its full length. Among the more common impediments are narrowing of lumen, or fixation of bowel from without by neoplastic or inflammatory disease, danger of provoking hemorrhage or perforation in an acutely inflamed zone, unusually prominent sacral promontory, and patient's intolerance of the procedure.

2. Status of mucous membrane. Injection, edema, bleeding, or ulceration may be evident. The *location* and appearance of any local lesion and the appearance of the mucous membrane below and (if possible) above it should be noted

3. Blood This may be apparent at once or found only on examination of material *wiped out*. (Wipings should always be carefully inspected and, under pertinent circumstances, saved for microscopic examination and perhaps culture) It is important to observe whether blood is localized in the lower bowel or comes from a point higher up Occasionally the instrument, as it is inserted, will carry blood from the anal ring upward or the mild trauma incident to its passage will create slight bleeding from a normal mucosa. Careful scrutiny of the membrane beyond the tip of the sigmoidoscope as it advances will enable one to blame or exclude either of these eventualities Sometimes the only indication of a benign adenomatous polyp of sigmoid or upper rectum will be a small amount of bloody mucus seen on the mucosa or oozing down from above.

4. Pus or mucopus.

5 Neoplastic growth.

Fully as important as inspection of the wall during introduction of the sigmoidoscope is inspection during withdrawal. This pertains to the valves particularly Each valve should be carefully "ironed out" by manipulation of the instrument so that its upper as well as its lower surface can be seen Otherwise, a small polyp or carcinoma starting on an upper surface will be missed

Disorders of Anus and Rectum

Pruritus Ani. Intractable itching of the anal mucocutaneous area, often accompanied by burning, can be created by a wide variety of local causes including low-grade infection traceable to uncleanliness, dermatophytosis, discharge from the rectum or a neighboring part, hemorrhoid, fissure, pinworms or other parasite, diarrhea, sensitivity to toilet tissue or some local application, or syphilitic condylomas It can also occur in uremia, diabetes, jaundice, allergic states, and other systemic disorders causing itching elsewhere, in various dermatoses such as psoriasis, seborrheic dermatitis, neurodermatitis and eczema, and as one of the degenerative processes of advancing years It may accompany pruritus vulvae. An increasingly common cause is administration, especially by the oral route and for a long period, of various antibiotics; here destruction of normal intestinal flora and unrestrained growth of monilias, other fungi and perhaps antibiotic-resistant bacteria are thought to be at fault. Often intractable and therapy-resistant pruritus ani is encountered without demonstrable cause. The process may be confined to the anal region but often spreads to the perineum, scrotum, or vulva, and sometimes to the intergluteal folds, inguinal folds, and adjacent areas of the thighs

Occasionally no changes are evident on inspection As a rule the perianal skin will be grayish-white, boggy, thickened and perhaps show hypertrophy of folds, lichenification, fissuring, maceration, and moist secretion. Involvement of

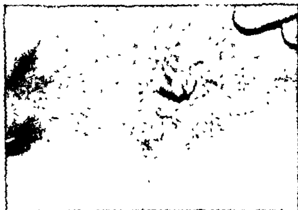


FIG 33.3 Pruritus ani, pronounced White, lichenified perianal skin, hypertrophied folds, and ulcerations presumably secondary to infection created by scratching Process extends to intergluteal fold and perineum (Courtesy Dr E Parker Hayden)

the perineum and vulva is indicated by reddening, puffiness, and excoriation, followed by whitening, thickening, and fissuring. The scrotum, intergluteal or inguinal folds or adjacent areas of the thighs may show redness, scaling, and excoriation, in the folds, bogginess, maceration, and fissuring are also likely

Hemorrhoids. EXTERNAL These are dilated veins visible at the external orifice They occur singly or multiply and, unless thrombosed, usually give no symptoms other than perhaps itching and irritation reflecting low grade inflammation secondary to difficult cleansing of the area Thrombosis causes local pain—increased on sitting, walking or defecation—itching, acute tenderness, and increased swelling which may involve the surrounding tissue The overlying skin may break, resulting in extravasation of fresh and clotted blood. Spontaneous gradual healing is the rule; the end result is a small tab of fibrous tissue covered with skin

INTERNAL Here the dilated veins originate around the internal orifice They may cause no trouble but, as a rule, periodic bleeding occurs, especially at defecation A probable diagnosis can be made by digital examination provided one can feel thickened longitudinal folds in the anal canal. A positive diagnosis can be made only by seeing the swollen, bluish-red folds through the anoscope. An internal hemorrhoid often gradually enlarges downward along the canal and may eventually protrude, particularly during defecation, it is seen on external examination as a bright red or purplish-red, spongy mass which bleeds easily Although it may recede spontaneously or with the aid of external pressure and manipulation, it is likely to reappear following defecation or straining. Strangulation or thrombosis may occur causing intense pain and itching.

Two important facts to remember in connection with hemorrhoids are:

1 *Bleeding from the rectum due to carcinoma is often carelessly and incorrectly attributed to existent or non-existent internal hemorrhoids.*

2 *Unnoticed or discounted loss of blood over a long period may be the cause of obscure anemia.*



FIG 33 4 Large external and protruding internal (head of arrow) hemorrhoids (Courtesy Dr. F Parker Hayden)

Fecal Impaction. A large hard mass of inspissated fecal material collects in the rectum This is a common, yet frequently overlooked, cause of obstipation and distention It can develop as a result of neglect of bowel hygiene, inability to defecate because of local pain or some neuromuscular disorder, recent ingestion of a barium x-ray meal, or generous use of opiates or certain antacid and antidiarrheal agents. Often it is encountered in bedridden, severely ill, postoperative, or stuporous patients It can be avoided by day-to-day checks of bowel status and repeated rectal examinations.

The patient is apt to complain of flatulence, abdominal discomfort, fulness or cramps, constant urge to defecate, or a feeling of "something in the rectum" Oral catharsis will cause diarrhea but as a rule will not effect expulsion of the mass unless it is first broken up by digital manipulation and local instillations.

Fissure in Ano. A small crack or area of ulceration in the mucosa of the canal occurs at a commissure, occasionally elsewhere Severe pain and reflex spasm of the sphincter on defecation, and slight bleeding with the movement are the chief manifestations The patient often becomes constipated because of inability to defecate due to pain and spasm The fissure can be seen on gentle separation of the mucosal folds, or on inspection through a small anoscope Digital examination or introduction of a large instrument is often impossible because of pain

Fistula in Ano. Infection spreads into the perianal tissues from a minor lesion in the anal canal or, less often, the rectum, such as a fissure, hemorrhoid or abrasion from a fishbone or other sharp object in the feces As a rule, it works its way through to the outside so that *true* fistula actually represents a minute channel with one opening inside the anus or rectum and the other through the skin near the anal orifice. Tuberculosis is an occasional cause Sometimes the process begins on the outside from infection of a hair follicle and extends upward The channel is tortuous and may have multiple openings. It opens and closes periodically; when active it causes slight local discomfort and usually

external fecal-contaminated discharge. On examination, one finds near the anal orifice, a pinhead opening surrounded by a narrow zone of induration The internal opening can rarely be seen but a small zone of induration and tenderness may be felt digitally.

If, as occasionally happens, infection starting within does not burrow through to the outside, a so-called *blind* fistula is created. Some degree of perianal pain is the rule; intermittent staining from seepage through the anal ring may occur Here, too, digital palpation may reveal slight thickening and tenderness.

In either type, if the process becomes sufficiently active and drainage is impaired, ischiorectal abscess can develop

Ischiorectal Abscess. If an area of active perianal infection from one of the causes just noted fails to drain adequately, pus collects in the ischiorectal fossa The classic indications of abscess are present. local pain, constitutional symptoms, fever, and leukocytosis When the lesion points externally, redness, tenderness, and swelling are found in an area near the anal orifice. If it points higher up, it can be felt on digital examination as a tender, local, inward bulging of the rectal or anal wall In a doubtful case, one might be able to detect tenderness and perhaps induration with his forefinger in the rectum and his thumb pressing toward it from the outside Following spontaneous or surgical drainage, a fistula may remain

Diffuse Inflammatory Process. Changes in the rectum similar to those seen in the sigmoid and higher may be created by ulcerative colitis, bacillary dysentery, amebic dysentery and tuberculosis (*see* Chap 30).

Stricture of Rectum This can result from any ulcerative lesion such as tuberculosis, syphilis, ulcerative colitis, dysentery, gonorrhea or lymphogranuloma venereum, and from intrinsic cancer Luminal narrowing can also be created by pressure from without as from a large uterine fiomyoma or greatly enlarged prostate. Rarely, infiltration by an endometrial implant most likely at the rectosigmoid junction will be sufficient to cause some diminution of caliber. When due to an ulcerative lesion, diarrhea perhaps alternating with constipation is the rule; blood and pus are found in the stools When the process reaches the point of appreciable obstruction, constipation becomes more pronounced and the movements are thin, pencil-like, scant and frequent It must be remembered, however, that *diarrhea may predominate* as a result of repeated use of cathartics or passage of liquid feces around a solid mass obstructed above the lesion Rectal pain, discomfort, or sense of fulness, and sometimes, abdominal colic are the complaints Digital and sigmoidoscopic examination reveal a zone of narrowing, variable in length and with smooth or irregular walls (Carcinoma is described below.)

Pathologic examination of a specimen of tissue is often necessary to determine the nature of the lesion If a granulomatous process does not prove to be tuberculosis or carcinoma, a Frei test should be performed to exclude lymphogranuloma venereum.

Polyp. FIBROUS. A benign tumor probably representing the remains of a fibrosed hemorrhoid, fibrous polyp is found attached to the upper margin of the

anal canal. It causes no trouble other than some local discomfort if it is forced down into the canal during defecation.

ADENOMATOUS Adenomatous polyp is often multiple, appearing throughout the colon, and a likely cause of recurrent bleeding and excess mucus in the stools. Here too pain is a feature only if one is so situated that it will enter the canal during defecation. Occasionally one or more soft rounded masses can be felt by rectum. Through the sigmoidoscope, adenomatous polyps are visible as mulberry-like, relatively soft, multiple, polypoid growths, often attached by narrow pedicles They are most commonly found 15–20 cm. above the anus. Malignant change is a decided hazard.

Carcinoma of Rectum. The picture is similar to that of carcinoma elsewhere in the large bowel. Blood in the stools is usually the first *manifestation, although it is sometimes* preceded by lower abdominal or pelvic discomfort, gradually increasing constipation often alternating with diarrhea. "*Pencil*" *stools* sometimes occur but their diagnostic significance is overrated. Pus or mucopus may be passed The lesion can be felt as a firm, sometimes soft, nodular, elevated growth which partially obstructs the lumen Fixation occurs in the late stages In carcinoma of the anal canal, metastatic enlargement of the inguinal nodes may appear early. Through the sigmoidoscope one sees an irregular, elevated, ulcerated, bleeding lesion which partially narrows the lumen. If cancer is suspected—as it always should be whenever rectal bleeding or change of bowel habit occurs—and a lesion is not readily seen through the sigmoidoscope, the instrument must be manipulated so as to bring into view the upper surface of each rectal valve, a not infrequent site of early cancer Barium enema is often helpful, but a lesion may escape notice if it is small or situated low in the rectum *Negative x-ray studies do not exclude rectal cancer*

MALE GENITAL ORGANS

Disease of the male genitalia is suggested by a story of pain in the penis, testicles, or rectum, disturbances of micturition, hematuria, urethral discharge or bleeding, local irritation, inflammation or swelling, bloody ejaculation, or priapism Since some of these can reflect trouble elsewhere, their appearance does not necessarily indicate local disease Examination is made chiefly by inspection and palpation Further study of the prostate and urethra can be made by catheterization, panendoscopy and cystourethrograms.

The pubic hair must be examined, especially when the patient complains of local itching or irritation, for pediculosis (*see* Chap 4), and the skin of the genital area and adjacent surfaces of the thighs inspected for dermatophytosis (*tinea cruris*), and other cutaneous lesions Dermatophytosis appears as a sharply defined area of erythema, with a slightly elevated border composed of pinpoint vesicles

THE PENIS

Malformation. HYPOSPADIAS is a congenital deficiency of some portion of the ventral wall of the urethra often associated with a short, downward curved penis



FIG. 33 5 Chancre of penis.

EPISPADIAS, a rarer variant, is deficiency of the dorsal wall occasionally accompanied by exstrophy of the bladder

Phimosis. Because of a narrow orifice or adhesions between its inner surface and the glans, the prepuce cannot be retracted An associated balanitis is likely to occur as a result of necessarily inadequate cleansing, gonorrhea, or other local infection

Paraphimosis. The prepuce becomes caught behind the corona and cannot be brought forward Resulting impairment of local circulation to the glans results in edema and, if the situation is not relieved, gangrene

Balanitis. Inflammation of the glans is most apt to occur in association with phimosis Poor hygiene, gonorrhea, and injudicious use of irritating local applications are the most common causes The usual signs of inflammation are found, ulceration may develop Inguinal nodes can become swollen and tender but rarely suppurate In the presence of phimosis, an inflammatory lesion of the glans may require dorsal slit or preferably circumcision to exclude chancre, carcinoma, or other serious lesion

Priapism. Prolonged erection, usually painful, and not related to sexual desire, is seen in spinal cord disease or injury, chronic myelogenous leukemia, thrombosis or tumor of the corpora cavernosa penis, and sometimes in acute prostatic infection

Syphilis Chancre is a superficial, painless, indolent ulcer with an indurated base and a scanty serous discharge Rarely, it is multiple Its commonest sites are the glans and inner surface of the prepuce Moderate hard, painless, nonsuppurating swelling of the inguinal lymphnodes is usually present Diagnosis depends on demonstration of *Treponema pallidum* by dark-field examination of tissue juices squeezed from the lesion This procedure can be regarded as negative only if the organisms cannot be found on repeated examinations. If the dark-field test is positive, a positive serologic response is confirmatory If the former is repeatedly negative, a positive serologic reaction can be regarded as diagnostic in the appropriate case Since the blood test may not become posi-

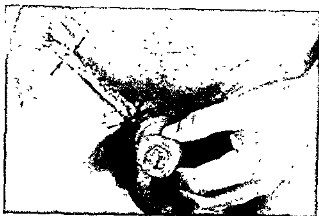


FIG 33.6 Chancroid Ulceration and edema of prepuce with phimosis Arrows indicate line of incision where suppurating inguinal lymphnodes were surgically drained (Courtesy Dr Earl A. Glicklich)

tive for some time following exposure, an early negative finding is by no means conclusive, repeated examinations must be performed for at least three months. *In recent years, the dependability of these criteria has been greatly reduced by antibiotics* If, shortly before or for some time following exposure, penicillin or other antibiotic has been applied locally or administered systemically for prophylactic purposes, or for treatment of obvious gonorrhea, other local infection, or some totally unrelated disturbance, chancre may not develop in its usual form, and dark-field studies are apt to be persistently negative. Furthermore development of a positive serologic reaction may be appreciably delayed;

Chancroid. A localized venereal disease transmitted only by direct contact, this is endemic primarily among peoples who live at a low hygienic level The lesion, occasionally single, usually multiple, begins as a vesicopustule but rapidly breaks *down to form a painful, round, or irregular, superficial or, sometimes*, deep ulcer with an irregular, grayish-yellow base which discharges profuse, yellow, purulent material and bleeds easily if traumatized Surrounding it are areas of redness *without induration The initial sore appears in the pocket* on either side of the frenum, in the coronary sulcus, on the shaft or sometimes the anal mucocutaneous area Autoinoculation is common In about one third of cases, inguinal lymphnodes become large, *tender and sometimes suppurate* Diagnosis may be confirmed by finding the organism in stained smears or cultures taken from a lesion or involved lymphnode The picture is confused by previous administration of a sulfa drug *or other antibiotic, except penicillin. Since the lesions of chancroid are likely to conceal a coexistent chancre, the presence of syphilis must always be suspected, especially if specific prophylaxis or therapy has been given.*

Granuloma Inguinale. Known also as *granuloma venereum*, this disease predominates in the Negro race in this country. Although some doubt exists con-

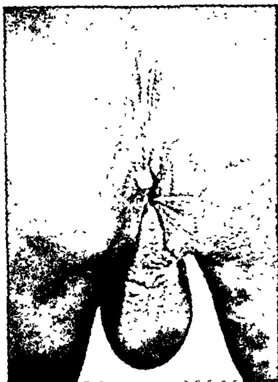


FIG 33 7 Granuloma inguinale Ulceration and granulation of perianal region, perineum, and posterior aspect of scrotum Anal canal constricted by extension of process upward into its wall, orifice (tip of arrow) incompletely closed because of cicatricial rigidity (Courtesy Dr Earl A Glicklich)

cerning its manner of spread, it is generally regarded as a venereal infection of low communicability It begins as a sharply defined papule, vesicle or nodule on the skin of the inguinal or genital region, on the glans, or inner surface of the prepuce. It is usually painless and soon develops into a slowly growing, indolent ulcer which spreads peripherally Its base is reddish, soft, exudes scanty gelatinous material, and bleeds easily following trauma The edges are irregular, turn out, and overlap the bordering epithelial surface A lesion may heal with scar formation along part of its edge and progress at another, cicatrization in a healed segment may be cartilaginously hard Spread by contiguity is common, new lesions being most likely to appear on moist surfaces such as the scrotum, and inguinal or intergluteal folds Regional nodes are perhaps slightly enlarged but do not become tender or suppurate Lymph stasis can create enlargement of penis, scrotum, or labia Eventually impairment of general health may occur, sometimes with involvement of bones, joints, and viscera. Diagnosis is confirmed by demonstration of large mononuclear cells containing Donovan inclusion bodies in scrapings or punch-biopsy specimen obtained from a clean but active segment of a lesion



FIG 33 8 Lymphogranuloma venereum Arrow points to small primary lesion Homolateral inguinal nodes enlarged and fluctuant (Courtesy Dr Earl A Glicklich)

Lymphogranuloma Venereum. Also called *lymphopathia venereum* and *lymphogranuloma inguinale*, this is a systemic virus disease acquired venereally It begins as a small, transitory, painless, papular, herpetic or ulcerative lesion of a genital organ, the perianal area, or the anus, often so inconspicuous as to escape notice Within 10–30 days following infection, inguinal lymphnodes uni- or bilaterally become enlarged, tender, and adherent to the skin, this is often the first obvious manifestation The skin overlying the involved nodes may show horizontal creasing or fissuring and acquire a dusky or purplish hue Later the nodes become fluctuant, matted together, and eventually break down with formation of multiple draining sinuses followed by partial healing, scar formation and puckering. During this phase constitutional symptoms—fever, malaise, weakness, loss of weight and sometimes moderate leukocytosis—are the rule In some cases the patient develops a cutaneous eruption, myalgia, arthralgia, conjunctivitis, or indications of low-grade meningitis Impairment of local lymphatic circulation can create elephantiasis and deformity of the genitals, with perhaps abscesses and sinuses based on secondary infection

In the female, the primary lesion is rarely evident. Owing to the anatomic difference in lymph drainage, involvement of inguinal nodes is less common than in the male but a granulomatous process in the rectum is likely Often the first indication of the disease, this is marked by bleeding, purulent discharge and, if untreated, eventual development of a stricture (In the male, rectal involvement is less common and by some observers thought to occur only if the primary lesion is in the anus, rectum, or perianal region.) Occasionally disease of the vulva and adjacent parts results in granulomatous changes, edema, ulceration, and gross deformity of these structures (*esthiomene*)

The diagnosis of lymphogranuloma venereum is confirmed by a positive Frei test.

Herpes. Herpes of the penis is common On the shaft the lesion is similar to that found elsewhere. On the glans beneath the prepuce it may ulcerate and be mistaken for chancre or some other serious process. It is often a cause of unwarranted concern to the patient.

Sebaceous Cyst. Another common source of apprehension to the patient is his discovery of one or more small (1–3 mm.) nodules in the skin of the penis or scrotum. On palpation sebaceous cyst, which is benign, is non-tender unless secondarily infected, firm, circumscribed, freely movable, and obviously confined to the skin and subcutaneous tissue

Papilloma. Also called *condyloma acuminatum* and *venereal wart* this is a benign, warty overgrowth of tissue, usually multiple Favorite sites are the glans, coronal sulcus, scrotum, and perianal skin Presumably an infectious process, it is likely to be related to uncleanliness, and often seen following balanitis or urethritis

Epidermoid Carcinoma. The lesion is usually found on the glans, coronal sulcus, or prepuce, less often the shaft The first three are rarely, if ever, the site of carcinoma if circumcision was performed in early life Typically, the picture is of a verrucous excrescence or an infiltrative, ulcerative growth having the usual characteristics of epidermoid carcinoma elsewhere. When seen early on the glans or corona, it may appear as a thickened leukoplakia-like area, any such lesion should be regarded as carcinoma until proved otherwise The inguinal lymphnodes are not involved until late, they are then hard and shotty but may be soft if secondary infection is present Carcinoma can be readily mistaken for chancre or other granulomatous or ulcerative lesion In the doubtful case, biopsy is essential When the prepuce is not retractable, pain, swelling, tenderness, or other indication of trouble beneath it cannot, as a rule, be evalu-

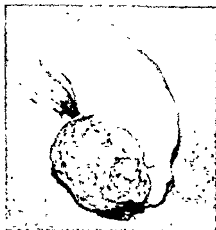


FIG 33 9 Epidermoid carcinoma, extensive, arising on lip of prepuce. (Courtesy Dr Wyland F Leadbetter)

ated without dorsal slit or circumcision, so that the lesion can be properly examined and perhaps biopsied.

Urethritis. Gonorrhea accounts for most of the cases and produces the most severe form

ACUTE GONORRHEAL URETHRITIS is marked by local pain and burning on micturition, and purulent urethral discharge appearing 3–5 days after contact, and often accompanied by inflammation of the prepuce and glans. Formerly, the disease was frequently marked by extension of infection to the posterior urethra, prostate, vesicles, and epididymis, and sometimes by systemic spread, especially with development of gonorrheal arthritis in one or more joints Modern therapy has greatly reduced the incidence of these complications.

CHRONIC GONORRHEAL URETHRITIS Probably a reflection of smouldering infection in the prostate or vesicles, this was common before introduction of modern therapy but is now less frequent. Usually asymptomatic, it is evidenced by a scanty discharge or, when this is not obvious, by gluing together of the meatal lips in the morning. The diagnosis of gonorrheal urethritis is established by demonstration of the organism by direct smear or culture of the secretion.

NON-SPECIFIC URETHRITIS This is caused by other organisms, including the trichomonas Trauma—usually instrumentation or injection of a strong antiseptic—alcoholic or sexual excess, or occasionally excretion in the urine of some irritating substance such as cantharides may be a contributing factor Some cases represent extension of infection from the prostate The picture, as a rule, is similar to, but less severe than gonorrheal urethritis, antibiotic therapy is apt to be less effective.

A syndrome of unknown etiology characterized by conjunctivitis, urethritis, and arthritis is sometimes encountered (*Reiter's syndrome*) (*see* Chap 35)

Periurethral Abscess. This is a complication of gonorrheal or non-specific urethritis. It is often seen secondary to infection resulting from an inlying catheter or repeated catheterization, especially in paraplegia and other central nervous system disorders in which the mechanism of urination is impaired The abscess may vary from a small tender swelling discovered by palpation of the ventral surface of the penis along the course of the urethra, to a large, tense, painful mass involving most of the penis and scrotum. Systemic indications of infection are present Stricture formation due to scar tissue contraction associated with healing is a common sequela Once a stricture has formed, chronic infection persisting proximal to it may create additional abscesses and strictures Urethral fistula may result from external rupture of an abscess, urine will then be voided through the abnormal opening as well as through the normal channel

Urethral Stricture. Usually representing the end result of periurethral abscess, this creates diminution in size of the stream, increasing difficulty in voiding, and later, indications of chronic retention. Secondary obstructive changes in the bladder, including stone and diverticulum are common, in the long-standing case, trouble reflecting back pressure in the kidneys may develop. Complete retention is likely. Diagnosis is made by passage of sounds, and panendoscopy.

The Testes and Scrotum

The scrotal contents are palpated between the thumb and fingers. Normally the testis is felt as a sensitive, smooth, firm, ovoid structure, freely movable within the scrotum. At its upper and lower poles and extending between them along its posterolateral margin, one can identify the softer, irregular cord-like epididymis. From the level of the upper pole upward to the external inguinal ring, the spermatic cord is felt as a relatively soft, roughly pencil-sized cord-like structure within which one can roll between the examining thumb and finger the much firmer, smoother, smaller (1–2 mm diameter) vas deferens. Important to note are size, shape, firmness, increased tenderness of these structures, and alteration of normal subjective testicular sensation on pressure. Corresponding parts on the two sides must always be compared In the presence of pronounced edema, large hydrocele, or large scrotal hernia, palpation of homolateral scrotal contents is often unsatisfactory.

Edema. Edema of the scrotum and penis occurs in any acute local inflammatory process, in association with general or dependent edema in such disturbances as nephrosis and congestive heart failure, and obstruction to flow of blood or lymph from any cause such as thrombosis of vena cava, malignant disease of inguinal nodes, and lymphogranuloma venereum In endemic areas, filarial infection produces huge swelling of the genitals When edema of the penis is pronounced, swelling and downward extension of the prepuce, except where it is anchored at the frenum, curves the organ in such a way as to create the so called "powder-horn" shape When scrotal edema is pronounced and predominant, the penis is almost encompassed and may be so concealed that visualization of the meatus and glans becomes extremely difficult

Hydrocele. An accumulation of serous fluid in the tunica vaginalis can occur acutely from trauma, inflammation, such as epididymitis or orchitis, or testicular tumor A chronic slowly developing form of unknown cause is more common. Sometimes hydrocele is a congenital variant, here it is likely to communicate with the peritoneal cavity and may change in size from time to time as the fluid flows back and forth Unless the hydrocele is secondary to an acute inflammatory process, it is asymptomatic except for a local dragging sensation One finds a smooth, tense, pear-shaped, non- or slightly fluctuant, usually unilateral swelling, pain and tenderness occur when there is an associated inflammatory process As a rule, the testis and epididymis can be palpated behind the swelling and normal spermatic cord above it but a large collection of fluid may conceal the first two Except when a complicating hernia exists, the percussion note is dull and no impulse is obtained on cough Hydrocele is usually translucent; if in a dark room, a flashlight is held against the affected side, the hemiscrotum will be suffused with a pinkish glow, within it a dark shadow representing testicle may be perceived Light is not transmitted if the fluid is opaque or bloody, or the tunica thickened Diagnosis can be established by exploratory puncture but this procedure must never be attempted unless one can be sure that the swelling is not due to hernia or tumor Following puncture-drainage

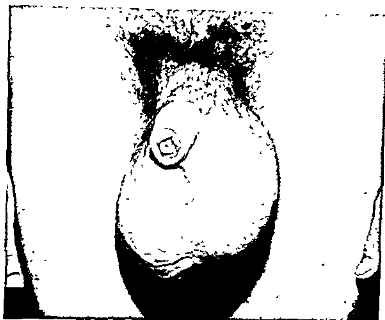


Fig 33 10 Swelling of scrotum due to bilateral hydrocele

of a hydrocele, the scrotal contents must be carefully palpated to exclude an underlying inflammatory or neoplastic lesion

Hematocele. Usually following trauma, blood in the tunica vaginalis creates a firm, non-translucent, non-fluctuant mass closely resembling tumor To distinguish it from the latter, exploration is often necessary This is true even when a history of recent trauma is obtained, often the patient will first detect testicular enlargement which is actually due to tumor by self-palpation motivated by pain or tenderness secondary to the injury.

Spermatocele. A collection of milky fluid containing spermatozoa is confined in one or more cysts arising from the epididymis Spermatocele resembles hydrocele but can sometimes be differentiated by determining on careful palpation that it arises from the upper pole of the epididymis. Puncture or exploration is often necessary for positive diagnosis

Varicocele. Multiple varices of the pampiniform plexus usually encountered on the left and due to valvular insufficiency of the spermatic vein are recognized as a mass of tortuous veins palpable in the scrotum A dragging sensation in the scrotum or groin is the only symptom. Rarely, left varicocele is a complication of renal carcinoma based on impaired blood flow from tumor invasion of the spermatic or renal vein

Scrotal Hernia. In contrast to hydrocele, swelling of the scrotum due to hernia is tympanitic on percussion except when it is composed chiefly of omentum, is non-translucent, creates a palpable impulse on cough and, as a rule, can be made to disappear by manipulating the contents of the sac back into the peritoneal cavity In the doubtful case, history of gradual progress of the swelling from above downward is helpful The testis and scrotum may be

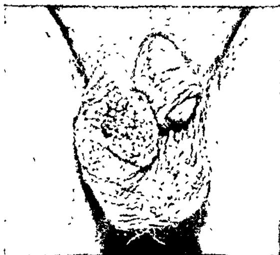


Fig 33 11 Epidermoid carcinoma of scrotum Patient's occupation in a rope processing establishment was such that his clothes were constantly saturated with a material containing coal-tar derivatives. (Courtesy Dr Wyland F Leadbetter)

palpable posteriorly or laterally Auscultation is sometimes a helpful procedure; hernia is almost certain if, with his stethoscope receiver placed against the scrotal swelling, the examiner can hear peristaltic sounds X-ray may show a collection of gas or, following a barium meal, barium in the scrotal area. Scrotal *hernia must always be excluded before one punctures a swelling thought to be* hydrocele

Sebaceous Cyst. Occurring singly or multiply, this is *common in the skin of* the scrotum (*see* above)

Scrotal Carcinoma. Presenting the features of epidermoid carcinoma elsewhere, this metastasizes early to the femoral and inguinal nodes It is of relatively high incidence in chimney sweeps and those who work with coal tar products and lubricating oils, presumably because their work clothes are persistently saturated with the carcinogenic agent

Epididymitis. This is the most common infection of scrotal contents

Acute Epididymitis This was formerly, in a high percentage of cases, a complication of gonorrheal urethritis and prostatitis, but since the advent of modern antigonorrheal therapy, non-specific infection predominates The latter originates in the bladder, urethra, prostate or vesicles and is most likely to follow lower genito-urinary tract instrumentation or operation, or to occur as a complication of prostatitis which, in turn, arises as a complication of infection elsewhere, especially in the respiratory tract The epididymis is painful, swollen and acutely tender, *the scrotal skin, red and edematous Acute hydrocele* may develop Systemic response with fever, perhaps chills, and leukocytosis is usually pronounced. Often the patient will also experience pain in the homolateral groin and above it, due to involvement of the vas deferens (*vasitis*). In this event, tenderness and palpable swelling of the cord are likely. Sometimes in-

flammation of the vas within the inguinal canal or abdomen predominates; here, unless one also finds evidence of trouble intrascrotally, abdominal pain and tenderness may lead to a mistaken diagnosis of acute appendicitis or diverticulitis.

CHRONIC EPIDIDYMITIS. Usually secondary to chronic prostatitis or vesiculitis, this is marked by repeated attacks of acute epididymitis with intervening symptomless periods. During the latent intervals, the epididymis and cord will feel firm and thickened but are non-tender

TUBERCULOUS EPIDIDYMITIS. This is thought to arise from spread of infection from the prostate and seminal vesicles, which were previously infected by way of the kidneys. An active or latent extragenital focus in the lungs or elsewhere can almost invariably be found. Characteristically the disease is marked by gradually increasing swelling and pain within the scrotum The epididymis uni- or bilaterally is hard, irregular, nodular, enlarged and somewhat tender Associated involvement gives the vas a feeling of thickening and beading. In *the advanced case, fixation of the intrascrotal structures to the skin and* eventual development of one or more draining sinuses is likely. Spread to the testis is the rule (*see below*)

CONGESTIVE EPIDIDYMITIS Worth mention as another cause of unwarranted concern to the patient, this is a non-infectious process resulting from repeated or prolonged sexual stimulation without gratification It is marked by local aching or pain and tenderness of hours' to days' duration, and spontaneous recovery

Orchitis. ACUTE ORCHITIS This is likely to occur as an extension of severe acute epididymitis, especially when this appears after a genital tract operation It is common in mumps (*see below*), and, rarely, is encountered as a complication of typhoid fever or some pyogenic process elsewhere. The tender and swollen testis can usually be identified on palpation. The pyogenic form may proceed to abscess formation but this hazard has become relatively rare since introduction of antibiotics Abscess is to be suspected when the acute process fails to subside within a few days to a fortnight

MUMPS ORCHITIS Orchitis is a common complication of mumps, especially when the latter occurs beyond puberty Exquisite pain, tenderness, and symmetrical swelling of one or both testes begins 4–5 days to 1–2 weeks following appearance of the parotitis Particularly in epidemics orchitis can develop without evidence of mumps elsewhere Systemic reaction is often pronounced Epididymitis and hydrocele may occur *Although spontaneous recovery is the* rule, testicular atrophy is a decided hazard; some observers believe the latter can be prevented by surgical incision of the tunica immediately after the orchitis appears Sterility is a likely end result of bilateral atrophy.

TUBERCULOUS ORCHITIS Representing spread from the epididymis, tuberculous orchitis *cannot be detected* by local examination in the early stage but is often found in the surgically removed specimen. In the late case, testicular involvement can be assumed if one finds a large irregular mass and draining sinuses



FIG 33 12 Swelling of left side of scrotum due to mumps orchitis and epididymitis with associated secondary hydrocele (Courtesy Dr Louis Weinstein, Dept of Infectious Diseases, Massachusetts Memorial Hospitals)

CHRONIC ORCHITIS Most often due to syphilis, this is not common Generalized enlargement or atrophy may result from diffuse fibrosis. Or one may find a slowly growing firm non-tender nodule perhaps with generalized enlargement representing gumma, by local examination, this cannot be distinguished from tumor

Testicular Atrophy. Atrophy can be created by a variety of causes including mumps, syphilis, and certain other infectious processes, trauma, delayed descent, improperly treated torsion of the cord, impairment of circulation by contraction of scar tissue around the cord following herniorrhaphy, late portal cirrhosis of the liver, excessive exposure to radiation, intensive estrogenic therapy, and deficiency of the anterior pituitary, or adrenal glands. Whether the process is uni- or bilateral obviously depends on the cause The testicle and epididymis are small and sometimes lack normal sensitiveness Such factors as hypoplasia of other sexual structures, diminution or absence of secondary sexual characteristics, impotence, and lack of fertility are dependent on the nature and extent of the underlying trouble, and, in certain instances, the age of onset

Cryptorchism. Arrested descent accounts for almost all cases of absence of a testis from the scrotum. The variant can be uni- or bilateral. It is usually possible to palpate the undescended testis within the inguinal canal or in the groin near the external ring, associated congenital hernia is the rule. The patient is likely to complain of local pain and tenderness, a reflection of pressure from surrounding muscles Atrophy is frequent Sometimes descent is so completely arrested that the testicle is retained in the abdomen and consequently is not palpable. Malignant disease is more common in the undescended than in the normal organ

Malignant Tumor. This takes the form of embryonal *carcinoma, seminoma,* chorioepithelioma, or mixed tumor (*teratoma*). The type can be determined only by microscopic examination. The testis is enlarged, smooth or nodular, *hard or soft, and in the early* stage, usually painless and non-tender. However, following trauma even of a minor nature, temporary pain or tenderness may make the patient aware of something wrong, whereupon palpation by himself or his physician leads to the discovery of the enlargement. For this reason, the development of malignant disease is often incorrectly attributed to local injury. Later, *a dragging sensation in the scrotum* or groin, obvious swelling, and perhaps thickening of the cord due to upward extension of the process will be evident. Since the lymph drainage is to the para-aortic nodes, these are the first focus of local metastasis. Their enlargement is often enough to cause displacement and perhaps obstruction of the homolateral ureter. Involvement of inguinal nodes and skin of the scrotum is a late *complication*. *The disease is* usually highly malignant and results in early death from metastasis to liver and lungs. Sometimes a metastatic lesion is the first indication of trouble. In a few patients, the tumor produces hormones which cause pain and swelling of the breasts and perhaps a positive Aschheim-Zondek test.

Torsion of Spermatic Cord. Because of congenitally inadequate fixation of the epididymis to the tunica, the testis can spontaneously revolve in its sac and thus create a twist and consequent vascular obstruction of the cord. It is *characterized by sudden onset of acute pain in the testicle and cord, edema of* the scrotum, and swelling and exquisite tenderness of the testis and epididymis. Torsion should always be suspected in any patient with acute intrascrotal pain, particularly if it begins abruptly and there is no evident cause for epididymitis such as an infected prostate. Sometimes the twist will spontaneously abate or can *be relieved by manipulation,* otherwise immediate surgical interference is essential. If the insult is not promptly corrected, gangrene of testis, epididymis, and the cord below the point of torsion will develop. Testicular atrophy is a likely end-result. It is possible that the occasionally encountered patient who complains of intermittent attacks of testicular pain may be having repeated mild and spontaneously subsiding episodes of torsion.

The Prostate Gland

Palpation of the prostate gland through the rectum is best accomplished with the patient in the knee-chest position, or standing with his trunk bent forward as nearly parallel to the floor as possible. Feeling chiefly the posterior and superior surfaces, one obtains the impression of a *smooth, rounded, globular,* roughly horse-chestnut sized, firm but somewhat resilient structure. As a rule, a sagittal groove is apparent on the superior surface and upper portion of the posterior, this represents the sulcus between the lateral lobes. By pressing medially on the lateral borders, slight side-to-side mobility can be detected. *The gland is small and* undeveloped prior to puberty while beyond mid-life it becomes larger than during the intervening period. Usually, the normal vesicles are not palpable but if distended with secretion they may, by the trained finger,

be identified as small ovoid structures softer than the gland, and extending obliquely upward and laterally from its superior surface

In palpating the prostate one feels for variations of size, consistency, sensitivity, symmetry, and mobility, and for irregularity or nodularity As in so many other instances, knowledge of what constitutes normal can be gained only by experience. Examination of the gland is not complete without microscopic study of its secretion which can be obtained from the urethral meatus following brisk digital massage.

Prostatitis. ACUTE PROSTATITIS. This is brought about by an infectious process elsewhere in the genito-urinary tract, such as urethritis, cystitis, or pyelonephritis, and almost equally commonly by infection elsewhere, particularly in the upper or lower respiratory tract. Sometimes the source cannot be determined. Formerly gonorrheal urethritis was the most common underlying disease but since the advent of modern therapy, other causes now predominate

Onset is heralded by pain in the sacral region, penis, rectum, or perineum, frequency and urgency of micturition, sometimes with burning or strangury, and possibly acute retention Defecation may be painful, and priapism occasionally develops Rarely, the patient will note mild hematuria Fever, leukocytosis, and other systemic responses are the rule On palpation the gland is acutely tender and homogeneously swollen The urine and prostatic secretion obtained by massage contain pus. If prostatic abscess forms, one or more of the above manifestations will become more severe The gland is exquisitely tender and one lobe usually more tender and swollen than the other

CHRONIC PROSTATITIS Resulting from any of the causes listed above, chronic prostatitis may be asymptomatic or cause local symptoms similar to but less pronounced than those of acute prostatitis Rarely, bloody ejaculation is the first hint of trouble To the examining finger the gland may feel normal or somewhat enlarged and either soft or firm Diagnosis is established by discovery of pus in the prostatic secretion or, if it was not present beforehand, pus in urine voided immediately following prostatic massage. *Chronic prostatitis must always be excluded by examination of prostatic secretion and/or post-massage urine specimen whenever the patient is being searched for a focus of infection, which might be related to some other disturbance such as arthritis or iritis.* This statement is also applicable to the man who feels below par, since chronic prostatitis, usually without local symptoms, is a common cause of easy fatigability and other non-specific complaints It must also be emphasized that the secretion may appear normal following one massage but will be found to contain pus if the procedure is repeated a day or so later.

TUBERCULOUS PROSTATITIS. This is not to be regarded as a separate entity but as one of the manifestations of genito-urinary tract tuberculosis. The initial genito-urinary lesion is usually in the kidney; the prostate presumably becomes infected by way of the bladder There are no distinguishing symptoms of prostatic involvement It is rarely suspected unless accompanying tuberculosis of the bladder or epididymis is found The gland may feel normal or be uni- or bilaterally irregular and somewhat nodular, occasionally it is definitely en-

larged, hard, and indistinguishable from carcinoma. Diagnosis may be established by finding tubercle bacilli in the secretion by direct examination, culture, or guinea pig inoculation.

Prostatic Calculi. Calculi in the prostate are not uncommon They may be found in a gland which is otherwise normal or in association with benign hypertrophy, carcinoma, or chronic infection Stones as such are usually asymptomatic. However, the patient is likely to have symptoms reflecting the associated lesion. The gland may be more firm than normal, sometimes it shows one or more areas so hard as to suggest carcinoma X-ray demonstration of calcium deposits in the prostatic region establishes diagnosis

Benign Hypertrophy. Common in men beyond middle life, this ordinarily is manifested by frequency and difficulty of micturition, nocturia, slowing of the stream, dribbling, and often, symptoms of urinary infection secondary to partial obstruction Total or terminal hematuria, or bloody ejaculation can occur. Acute retention may be superimposed at any time, occasionally it will appear without previous indication of trouble Sometimes incontinence representing overflow of a chronically distended bladder, or symptoms reflecting impaired renal function due to back pressure and infection from the distended bladder predominate. Rarely, enlargement is so pronounced that partial narrowing of the rectal lumen hampers defecation By rectum the gland feels enlarged, sometimes tremendously so, symmetrical, smooth, resilient, and not fixed The median sulcus may be exaggerated, or the opposite Catheterization shows residual urine, sometimes the catheter cannot be passed *It is not possible by rectal palpation alone to exclude prostatic hypertrophy as a cause of urethral obstruction* Most of the enlargement may project toward the bladder rather than posteriorly and hence not be appreciable on palpation Or a lesion of the middle lobe (which is not palpable) may be at fault In the absence of a recognizable enlargement, cystoscopy and other appropriate studies must be performed if symptoms suggesting impaired bladder emptying are present X-ray taken with opaque medium in the bladder will show elevation of its floor

Carcinoma of Prostate. The growth may be asymptomatic for a long time and remain undetected unless found in a routine examination or during a search for the primary focus of a metastatic lesion discovered in a bone, lung, or elsewhere Rarely, bloody ejaculation is the first sign of trouble In the early stage, since carcinoma almost always develops in the posterior part of the gland, digital palpation will show a small, firm to stony-hard, single nodule Later, evidence of obstruction similar to that of benign hypertrophy will appear This may be the initial indication of the disease Here one will find a large stony-hard nodule, or a slightly or greatly enlarged, irregularly contoured gland with loss of normal outlines and, if extension has occurred, fixation to the pelvic wall. By this time, pain in the sacral region or shooting down the legs is likely as a result of local invasion, nerve involvement, or perhaps extension into the lower spine. In addition to involvement of regional bones, widespread bone metastases, presumably by blood stream dissemination, are common Other late complications are invasion of the rectal wall, sometimes so extensive as to cause



FIG 33 13 Elevation of bladder floor by enlarged prostate Bladder filled with radiopaque medium 30 minutes after intravenous pyelogram

almost total obstruction, and of the base of the bladder and lower ureters with eventual hydronephrosis Occasionally metastases are found in the lungs or, less commonly, in the liver

If the nature of a nodule is not clear or one cannot distinguish between carcinoma, benign hypertrophy and tuberculosis as a cause of enlargement or obstruction, biopsy is essential

Except when prostatic carcinoma is undifferentiated, the acid phosphatase content of the blood serum is almost invariably elevated once the tumor has spread beyond the capsule In fact, an elevated acid phosphatase is virtually pathognomonic of prostatic carcinoma with extension, although its absence does not exclude the disease The osteoblastic activity associated with bone metastasis creates elevation of serum alkaline phosphatase.

Sarcoma of Prostate. Comparatively rare and more likely encountered in youth or early adulthood, sarcoma is a rapidly growing tumor which creates a tense, symmetrical swelling Usually symptoms of urinary obstruction appear first Local invasion and blood stream metastasis, especially to lungs and liver, are the rule



FIG 33 14 Carcinoma of prostate Mottled increased density of pelvic bones and femurs representing excessive osteoblastic activity of metastatic lesions

Seminal Vesicles

As indicated earlier, the normal seminal vesicles are not ordinarily palpable, although when distended with secretion they may be identified by the skilled observer as soft, sac-like structures extending obliquely upward and laterally from the superior surface of the prostate—one on each side of midline If infected, as they usually are in the presence of acute or chronic prostatitis, they are palpable when filled with purulent material In prostatic carcinoma, the vesicles usually become involved by upward extension of the disease and are then palpable as firm, cord-like projections above the gland.

THE FEMALE GENITAL ORGANS

To be complete, study of the female patient should include examination of the genital organs, but there are occasions on which one is justified in omitting this procedure No specific rule can be laid down except that it is better to be safe than sorry In virgins, vaginal examination should be omitted unless regarded as absolutely necessary (*see below*) Common symptoms requiring investigation are:

Local Itching or Irritation. Pruritus, burning, or irritation of the vulva, often

extending to the perineum, anus, vagina, and neighboring skin, can be due to a variety of causes, including pediculosis, dermatophytosis, poor hygiene, local infection, vaginal discharge, and, especially in older women, leukoplakia or senile vulvitis. Discomfort of this type is particularly common, usually from monilial infection, in diabetics and patients receiving intensive oral antibiotic therapy. At times local changes creating it are a reflection of a dermatosis or, as in itching due to jaundice, some systemic disturbance having cutaneous manifestations

Pain. Pain in the vulva may be caused by a local lesion, infection of Bartholin's glands, or be referred from the pelvis, rectum, or perirectal tissues. Pelvic pain or sensation of pressure or dragging, or low abdominal or back pain can be caused by numerous disorders, including salpingitis, ovarian cyst, malposition of uterus, extrauterine pregnancy, and tumor *One must not assume that every low backache is related to a pelvic organ.* It is more likely due to chronic fatigue, strain, or other musculoskeletal disorder of the low back, or it may be a toxic response of an acute or chronic febrile illness Sometimes pain originating in a pelvic organ is referred to the legs or, on defecation, to the rectum Whatever its character, it may be constant, intensified during catamenia, or occur only during catamenia

Dysmenorrhea. Low abdominal cramps beginning perhaps a day to a few hours before menstruation and persisting for 24–48 hours are a normal phenomenon in many women They are likely to be less pronounced following birth of the first child The term dysmenorrhea predicates more severe, sometimes incapacitating pain This is often attributable to idiopathic underdevelopment of the genital tract (*essential dysmenorrhea*) Other likely causes are pelvic inflammation, and occasionally malposition of the uterus or neoplasm of a pelvic organ (*acquired dysmenorrhea*) Pain continuing throughout the period suggests endometriosis or uterine malposition

Dyspareunia. In a high percentage of cases emotional factors are at fault Other possible causes are a vulval or vaginal inflammatory process, lack of secretion incident to aging, stenosis of the canal—congenital or acquired as from radiation therapy or chronic infection—and, occasionally, vaginal endometriomas If these factors can be excluded, one should suspect some intrapelvic disturbance, especially inflammatory disease, endometriosis, and ectopic pregnancy.

Change in Menstrual Cycle. Diminution or cessation of bleeding, excessive bleeding, or irregularity of periods suggests ovarian dysfunction, pregnancy—normal or abnormal—uterine polyp, or ovarian or uterine tumor. Scanty, absent, or infrequent menstruation is often a reflection of some systemic disease such as low grade infection, chronic fatigue, an endocrine disorder not confined to the ovary, or some emotional disturbance

Abnormal Uterine Bleeding. Bleeding without relation to menses is most likely due to ovarian dysfunction, miscarriage, or malignant disease of cervix, fundus, or ovary It can also be caused by estrogenic therapy

Leukorrhea. Mucopurulent discharge is usually the result of an infectious

process such as gonorrhea, trichomonas vaginalis, moniliasis, senile vaginitis, endocervicitis, cancer of the uterus, or overtreatment with some estrogenic product.

Inspection

The lithotomy position is preferable. The patient lies on her back with buttocks close to the edge of the table. When possible the feet should be suspended in stirrups. If the latter are not available, the legs and thighs should be flexed, the heels on the edge of the table as close as possible to the buttocks, and the knees widely separated. The bladder must be empty.

Vulva. After inspection of adjacent skin, hair, and perineum, the labia are separated, disclosing the clitoris, urethral orifice, vaginal orifice, hymen or hymenal remains, and outlets of Skene's and Bartholin's glands. Any lesion found should be palpated.

In virgins the hymen is a thin band of tissue stretching across the lower vagina, partially to almost totally closing the orifice. It shows an opening varying from pencil to finger size, sometimes there are multiple openings. In non-virgins, the hymenal remains can be seen as small tabs of tissue attached to the sides of the lower vagina. *In a virgin, examination should never proceed beyond the hymen unless it shows an opening large enough to admit one finger, or the circumstances of the case demand it.* The uterus and adnexa can be palpated through the rectum. If vaginal inspection and palpation are deemed necessary, especially in the apprehensive or tense patient, short-duration general anesthesia should be considered to spare her embarrassment, and to obtain enough relaxation for adequate examination.

Vagina. Prior to inspection and palpation, which require a lubricant, a specimen of secretion should be obtained for examination by the Papanicolaou method. Except when it is performed for some specific purpose as, for example, to follow the course of pregnancy, a gynecologic examination is not to be regarded as complete unless this test is done. It is highly useful in the diagnosis of early cancer of the pelvic organs, especially the cervix, but is dependable only if the interpretation is made by a person skilled in the field.

Inspection of the vagina should always precede palpation, otherwise the lubricant used on the gloved finger may, by clinging to the mucosa, conceal or be mistaken for mucopurulent discharge, or bruising of tissue during palpation might, by provoking bleeding, impair one's view. The Graves bivalve speculum is used, having first been warmed and lubricated. It is introduced gently into the vagina with the blades closed and vertical, when it is well beyond the orifice, it is turned so that the blades are horizontal, and then opened. By pointing the instrument in various directions and altering the depth of insertion, one can obtain a good view of the vaginal wall and the recesses around the cervix. Secretion or discharge can be removed by gentle wiping with gauze or cotton-tipped applicators. The normal vaginal mucosa is pink, smooth-textured, and somewhat moist, not unlike that of the mouth. One looks especially for variations of color

or moisture and for bulging, bleeding, discharge, areas of inflammation or ulceration, cysts, and tumor nodules

Cervix. The normal cervix, which varies in size depending on the age of the patient and her parity status, is normally seen as a cylindrical, somewhat tapered, roughly thumb-sized structure with its long axis extending downward and posteriorly and its free end pointing toward the posterior vaginal wall. Often, because of relaxation of supporting structures or uterine malposition, considerable manipulation of the speculum may be necessary before the cervix can be brought into view Its walls are covered by mucous membrane similar to that of the vagina The free inferior aspect is marked by thick, rounded, prominent lips that surround a somewhat sunken opening (*external os*) In the nulliparous the latter is oval, 1–3 mm in diameter, in the parous, slit-like, roughly 1 cm in length and irregularly edged. One looks for changes in the mucous membrane, laceration, bleeding or discharge, patches of leukoplakia, cyst, tumor, and ulceration In the hands of the specialist, colposcopic examination is sometimes helpful

Palpation

The left index finger is introduced slowly and with gentleness into the vagina, the back of the hand is pressed posteriorly toward the perineum to avoid contact with the sensitive clitoral region With his right hand, the examiner presses downward and inward on the abdominal wall starting at the level of the umbilicus and gradually moving the hand downward until the body of the uterus is felt (Strictly speaking, the *fundus* is that part of the uterus above the level of the entrances of the Fallopian tubes, the *body* the segment between this and the cervix In accordance with the custom in most clinics in this country, we use the two terms interchangeably to include both. The narrowed inferior end of the body where it merges with the cervix is known as the *uterocervical segment*.) By pressing simultaneously upward on the cervix and downward over the fundus, it is possible to outline the uterus, determine its mobility, and detect tenderness Obviously the cervix can be brought into better range of the intravaginal finger and hence be better palpated by downward pressure. Proper coordination of the hands is essential Obesity, abdominal distention, tenderness, or muscular tenseness due to pain or apprehension can seriously hamper adequate palpation Here, in the appropriate case, short-duration anesthesia is required

Following palpation of the body and cervix, the intravaginal finger is pushed upward in turn into the anterior and posterior cul-de-sacs and the lateral vaults, while the abdominal hand is pressed downward toward it When the lateral spaces are being palpated, the abdominal hand is moved from midline to the corresponding lower quadrant The normal findings are described below (*see Fornices*)

When vaginal examination has been completed, the forefinger is introduced into the rectum and bimanual palpation again performed. Previous findings may be confirmed, or variants discovered which were not detected by the vaginal

route. Only by rectum can one adequately feel the uterocervical segment. As noted above, rectal examination of the virgin often obviates the need for palpation through the vagina.

Vaginal Wall. Here one feels for degree of relaxation, areas of ulceration, thickening in the region of Bartholin's glands, nodules, and cysts

Cervix. The normal cervix is felt as a firm, cylindrical structure suspended in the vagina and pointing downward and backward; the rounded lips with the external os between them can be made out on the free inferior surface Points to be noted are the size, position, consistency, degree of mobility, and presence or absence of irregularity or nodularity

Body of Uterus. In general this is felt just behind the symphysis pubis as a smooth, rounded, firm, roughly pear-sized structure, freely movable between the examining fingers of the two hands It tends to be larger in women who have borne children and, irrespective of childbearing, smaller following menopause. The standards can be learned only by experience. It is normally in a position of anteversion and slight anteflexion with its long axis tending toward the horizontal, its anterior surface resting on the bladder, and its superior surface facing forward There is, however, considerable variation in different persons and also a range of change in the same person, depending on such factors as bodily position and the degree of distention of bladder and rectosigmoid To be noted are size, position, consistency, contour, and degree of fixation The size of the uterocervical segment should also be determined, widening indicates extension of malignant disease from the cervix or fundus into the base of the broad ligament Other points of importance are the degree of tension and consistency of the uterosacral ligaments, which extend backward from the level of the uterocervical segment toward the sacrum, a relaxed ligament is found in prolapse, nodular thickening most often in endometriosis

Fornices. Normally palpation of the anterior cul-de-sac reveals the fundus, but nothing of significance is felt posteriorly Laterally one may feel the sigmoid on the left or a low cecum on the right As a rule, the tubes and ovaries are not palpable but in a thin woman with lax abdominal wall either ovary may be detected as a movable, rounded, acorn-sized structure a few centimeters lateral to the fundus. Tenderness, fulness, downward bulging, thickening, fixation, or a mass indicate trouble.

Rectum. Best felt by rectum are posterior displacement or fixation of the uterus, tumor nodules on the pelvic floor or in the uterosacral ligament, and widening of the uterocervical segment.

Groin. Whenever inflammation, ulceration, or tumor of the external genitals is suspected, careful palpation of the groin for enlarged lymphnodes is essential They can be better detected with the legs extended or slightly flexed than with the patient in the lithotomy position

The Vulva

Inflammatory Lesions. The same lesions which appear on or near the male genitals may be seen in females, dermatophytosis, non-specific inflammation,

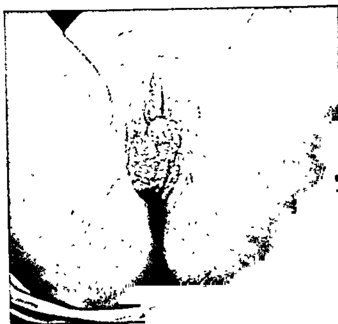


FIG 33 15 Condylomata acuminata of vulva and vaginal wall (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)

chancre, chancroid, lymphogranuloma venereum and granuloma inguinale In the female, chancre is likely to be multiple Condylomata acuminata and the condylomata of secondary syphilis appear in the labial folds, sometimes in the groin, and within the vagina

Discharge. Vaginal discharge is usually apparent on the external genitals, it causes redness and irritation of vulva and surrounding skin

Senile Atrophy. Following menopause, the clitoris and labia become smaller and their skin and mucous membrane thin, pale and dry Itching is likely Secondary infection may create mucoid or mucopurulent secretion Associated prolapse of the urethral mucosa is indicated by redness about the meatus The de-

[illegible] itches appearing [illegible] to occur following long-standing pruritus, especially when it is associated with senile atrophy. Redness, perhaps swelling, and cracking around the periphery of a patch indicate secondary infection (*leukoplakic vulvitis*) Although leukoplakia may appear around the anal orifice, the thickening and whiteness often observed here are more likely to be the end result of pruritus ani, not true leukoplakia

Kraurosis. Usually developing after menopause, this is regarded as a sequela of leukoplakic vulvitis. It is characterized by atrophy of the labia and clitoris, and dead-white, leathery, parchment-like skin which is often cracked, perhaps superficially ulcerated, and shows a sharp line of demarcation where it meets normal skin Itching may or may not be present

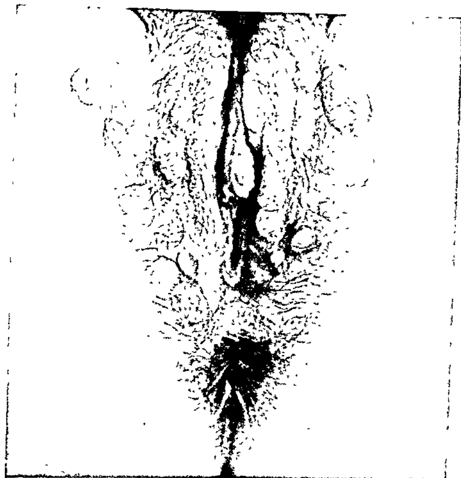


FIG 33 16 Syphilitic condylomata of vulva, perineum and adjacent skin

Carcinoma. *Often*, but not necessarily, superimposed on *leukoplakia* or kraurosis, cancer starts as an indurated nodule anywhere on the vulva, it may be flat or papillary, moist or dry Ulceration and early extension to inguinal nodes are the rule.

Varicose Veins. These are common during pregnancy and occasionally occur as a complication of a large pelvic tumor

Edema. This is often encountered as a feature of generalized edema Local causes include infection, pregnancy, large intrapelvic tumor, and lymphatic block due to carcinoma of the cervix or radiation therapy of the pelvis

Enlargement of Clitoris. *An endocrine disturbance* such as basophilic adenoma of pituitary, adrenal cortical tumor, or ovarian arrhenoblastoma is the most likely cause

Adhesions Between Clitoris and Prepuce. Usually accompanied by encrustations and pocketed secretions, these can be a source of local irritation and discomfort

Miscellaneous Lesions. Many patients become concerned by discovering some-



Fig 33 17 Carcinoma of vulva (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)

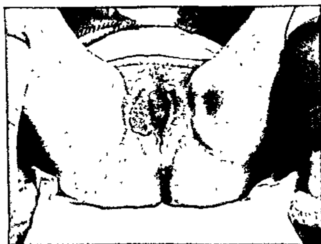


Fig 33 18 Extensive carcinoma of vulva (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)

where on the vulva an innocuous lesion such as a wen, lipoma, or nevus Other not uncommon sources of fright are vitiligo—patchy loss of pigmentation about the vulva, in the genital folds and on the inner aspects of the thighs—and melanosis—patchy darkening of the labia and adjacent structures. The latter is almost

invariably benign but if it is at all nodular malignant melanoma must be excluded

The Perineum

Laceration. Widening of the introitus often accompanied by appearance of the anterior vaginal wall in the orifice, loss of tissue elasticity, and thinning of the perineal body are frequently found subsequent to local injury during labor Although sometimes attributable to stretching alone, these changes usually imply tearing The various degrees of laceration and their end-results are:

First Degree. Only the vaginal mucosa was involved. No sequelae are evident.

Second Degree. The perineal muscles but not the rectal sphincter were torn. In addition to the signs just indicated, one finds scarring of the perineal body and some bulging of the posterior vaginal wall (*rectocele*).

Third Degree. Since the tear extended through the sphincter, the anal ring is relaxed and only a thin septum found between the vagina and rectum Local discomfort due to downward bulging of a rectocele is likely

Complete The rectal wall as well as the sphincter is torn Incontinence of gas and feces may be added to the manifestations of third degree laceration The sphincter and perineal body are absent, the rectal mucosa lies immediately adjacent to the posterior aspect of the vaginal introitus

Such symptoms as low pelvic discomfort, dragging, or heaviness, or low backache, if present, are more likely due to prolapse of uterus and adnexa than to the perineal injury itself.

The Vagina

Imperforate Hymen. This occasional variant results in retention of menstrual fluid beginning with the first period The hymen will be bluish in color and bulge downward If the situation is not corrected, retained menstrual blood and uterine secretions will distend the vagina (*hematocolpos*), and eventually the uterus and tubes An incompletely perforated hymen sometimes accounts for unsatisfactory coitus or sterility.

Vaginitis. Gonorrhea and trichomonas vaginalis are the most common causes of vaginitis and vaginal discharge

Gonorrhea In adults this involves the urethra, cervix, and Skene's and Bartholin's glands; the discharge originates from these structures Although the organism does not directly invade the vagina, its wall may show general or blotchy redness secondary to infection of the other structures In children primary involvement of the vagina is the rule The mucous membrane is intensely red, swollen, tender, and shows profuse purulent discharge

Trichomonas Vaginalis This is recognized by a blotchy mucosa and profuse yellowish-green discharge which frequently contains air bubbles and gives forth a characteristic pungent, acrid odor

Moniliasis This and other yeast infections actually involve the vulva more

than the vagina; they are particularly frequent in diabetics, and in persons receiving intensive oral antibiotic therapy The *secretion is watery, often containing white flecks of fibrinous exudate. Itching is intractable.*

SENILE VAGINITIS One observes redness, dryness and thinning of the mucosa, often with pinpoint spots of deeper redness representing submucosal vessels seen through the atrophic membrane Easy bleeding, mucoid discharge and perhaps ulceration may develop from secondary non specific infection acquired as a result of lowered local resistance

Bartholin's Glands. The small pinpoint openings of Bartholin's ducts can usually be identified in the normal woman, one on each side of the vaginal orifice just above the hymenal remains. Acute inflammation of the glands, most likely gonorrheal, is indicated by redness and pouting of the orifices but sometimes the latter cannot be seen because of concomitant inflammation of surrounding tissue *Often involvement will not become evident until long after the* manifestations of acute gonorrhea have subsided, at this stage, one will feel a local area of thickening in one or both posterolateral aspects of the vaginal wall Abscess of a Bartholin gland is characterized by exquisite pain and tenderness, purulent discharge, local swelling of the vaginal wall often sufficient to cause deviation to the opposite side. The entire vulval region may be red and swollen

Cystocele. Relaxation of the vaginal wall occurs as a result of age, or tearing or stretching of its supporting structures by childbirth *Cystocele is indicated by a downward, backward bulge usually as far down as the orifice It becomes more evident when the patient strains or stands In the equivocal case before cystocele can be excluded the patient must be examined while standing and straining (This statement also applies to rectocele and enterocele)* Incontinence, usually occurring only with sneeze, laugh, or cough, and frequency, urgency or other indication of urinary infection based on incomplete emptying are likely

Rectocele. Appearing as a downward, forward bulge of the posterior vaginal wall, this results from separation of the levator ani muscles and stretching of the vaginal wall If low, it will be seen at the vaginal orifice and become more pronounced with standing or straining If situated higher up, it can be best demonstrated with the examining finger in the rectum pressing downward and forward on the anterior rectal wall toward the thumb inserted in the vagina. If a rectocele becomes sacculated, local distress may be created by retention of fecal material and emptying of the pouch accomplished only by application of digital pressure from within the vagina.

Enterocele. A congenital herniation of peritoneum between the uterosacral ligaments, or an acquired hernia of the pouch of Douglas is felt as a downward bulge high in the vagina behind the cervix; when large, it is frequently confused with rectocele Here, too, examination is most successful with the patient standing and one's finger in the rectum and his thumb in the vagina.

Ulcerative Lesions. Non-specific inflammation, syphilis, chancroid, agranulocytic angina, and lymphogranuloma venereum are the more common causes of

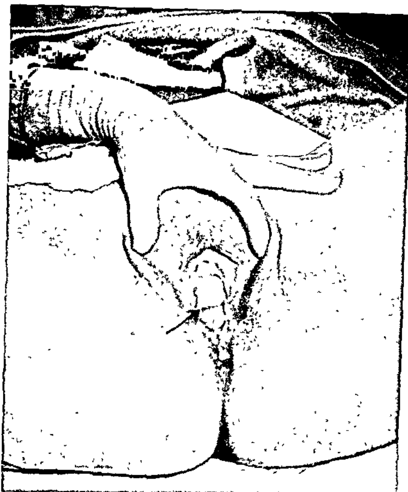


FIG 33 19 Lacerated perineum with cystocele and rectocele. Prolapsed segments of bladder and rectum fill vaginal orifice, which is represented by horizontal line (tip of arrow) Cystocele above, rectocele and lacerated perineum below (Courtesy Dr Joe V Meigs)

intravaginal ulcerative lesions Carcinoma must always be suspected when a *tender inflammatory or ulcerative process is encountered either within the* vagina or on the vulva or clitoris

Cysts. Multiple small cysts 0.5–10 mm in diameter are occasionally found on the lateral walls of the vagina. Vestiges of Gartner's ducts, these are of no significance except as an indication of lack of complete development.

Tumor. PRIMARY CARCINOMA. Primary carcinoma of the vagina or urethra is an indurated, hard, irregular 1- to 3-cm plaque which may or may not ulcerate, it must be considered in the presence of any *local inflammatory process*

METASTATIC CARCINOMA *This appears as small, friable, easily bleeding 0 1 mm. to 1-cm* lesions on the vaginal wall, they represent spread from the body of the uterus, ovary or, less commonly, the cervix

ENDOMETRIOMAS. Visible and palpable as small, bluish-red, firm nodules high

in the vaginal wall posterior to the cervix, these are apt to be overlooked unless the region is completely exposed to view by manipulation of the speculum. Except as an occasional cause of dyspareunia, vaginal endometriomas are asymptomatic. Their detection may lend support to a presumptive diagnosis of endometriosis within the pelvis.

The Urethra

Urethritis. The normal urethral orifice is flat and has a stellate opening. In acute cystitis, the urethra becomes inflamed; visible indications are minimal although one may find the orifice somewhat reddened and swollen. In acute gonorrheal urethritis, the meatus is definitely red, tender, swollen and exudes a thick, purulent, creamy discharge.

Skene's Glands. The orifices of Skene's glands, situated just inside the urethral meatus, are not ordinarily seen Because of atrophy of surrounding tissues, they are often exposed in women past the menopause, appearing as small, pinpoint openings on the posterior urethral wall In acute gonorrhea, they become widened, are usually visible, and discharge characteristic purulent material. In chronic gonorrheal infection pressure on either side of the meatus may force a small amount of pus out of the gland into the orifice. Occasionally the process will burrow upward in the periurethral tissues, creating a *periurethral abscess* which presents as a tender swelling along the lower third of the anterior vaginal wall

Urethral Prolapse. Usually the result of senile atrophy, the mucous membrane is everted, causing a red, non-tender ring of swelling about the meatus The pinpoint openings of Skene's glands may be visible

Urethrocele. Occurring independently or as an extension of cystocele, this presents as a longitudinal bulge along the anterior vaginal wall, usually with gaping of the urethral orifice

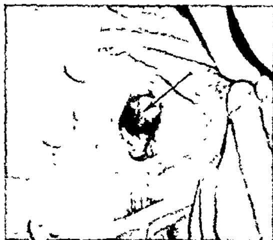


FIG 33 20 Large urethral caruncle (head of arrow) occupying anterior half of vaginal orifice (Courtesy Dr Wyland F Leadbetter)

Caruncle. A small vascular, bright red, polypoid lesion appears at the urethral opening, it is often exquisitely *tender and painful* during coitus and micturition Especially if such a lesion is hard or bleeds easily, carcinoma must be excluded.

THE CERVIX

As noted earlier, the normal cervix points obliquely downward toward the posterior vaginal wall. A cervix which is low means prolapse, one pointing directly downward or toward the anterior wall usually indicates *retroversion* or anteflexion

Bleeding. In a patient with abnormal bleeding, inspection will usually reveal whether the blood is coming from the cervix itself, as in inflammation, polyp, or malignant tumor, or from trouble higher up, such as endometrial tumor or hyperplasia, or miscarriage *Staining, especially after coitus or douching, is strongly suggestive of polyp or carcinoma of the cervix.*

Slight uterine bleeding occurring midway between periods, occasionally in some women, frequently in others, is most likely a phenomenon of normal ovulation

[illegible] as opposed to the definitely purulent, discharge usually seen in vaginal infection Gonorrhea and puerperal trauma of the cervix are the most common causes

Exposed Endocervical Epithelium. The cervix is covered by smooth, glistening, pink epithelium. Normally, none of the reddish-pink, bright endocervical mucous membrane is seen, but occasionally a symmetrical area of it is exposed, extending peripherally from the os for a distance of 1–2 cm. A congenital defect, this indicates lack of normal development, other manifestations such as infantile or juvenile uterus should be looked for It is frequently associated with dysmenorrhea and endometriosis Exposed endocervical epithelium of congenital origin is distinguished from that due to inflammation or tumor by its symmetrical distribution around the os

Laceration. When the cervix is torn at childbirth, it becomes asymmetrical, and endocervical epithelium is exposed The os is enlarged and extends through the cervical margin at one or more points, rents may be seen extending upward from it along the cervical wall. Mucopurulent discharge due to secondary infection is likely If lacerations heal spontaneously or are properly treated at the time of delivery, only scars are seen; exposed epithelium or discharge is not evident The injury is of importance only if the endocervix is exposed, or chronic infection present

Cervicitis. This is most likely due to gonorrhea, or injury and secondary infection resulting from abortion, childbirth, or instrumentation Hyperplasia of endocervical glands with secretion of mucopurulent material is the fundamental process Impaired drainage of the infected glands due to the infection itself, or to contraction of scar tissue secondary to previous trauma results in formation of retention cysts (*Nabothian cysts*), these appear near the junction of the cervical and endocervical epithelia as rounded, whitish prominences 1–3 mm



Fig 33 21 Prolapse and laceration of cervix Anterior lip held by tenaculum Black area is enlarged os Surrounding dark gray zone in which head of arrow lies is exposed endocervical epithelium (Courtesy Dr. Joe V Meigs)

or more in diameter Other possible complications of the long-standing case are polyp formation, obstruction of the cervical canal by granulations or scar tissue, and red inflamed areas on the cervical mucosa (*erosions*)

tending through the os will give a similar appearance Irregular bleeding, often precipitated by coitus or slight trauma is the important sign. In the presence of endocervicitis or uterine infection, mucopurulent discharge will be present. Polyp must always be differentiated from a pedulculated fibroid tumor presenting through the os, and from endocervical or endometrial carcinoma Sometimes this can be accomplished by inspection or Papanicolaou test but often curettage or other surgical procedure is necessary

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Slight uterine bleeding occurring midway between periods, occasionally in some women, frequently in others, is most likely a phenomenon of normal ovulation (*kleine regel*) It has no pathologic significance

Discharge. Infection of the cervical glands creates a mucoid or mucopurulent, as opposed to the definitely purulent, discharge usually seen in vaginal infection Gonorrhea and puerperal trauma of the cervix are the most common causes.

Exposed Endocervical Epithelium. The cervix is covered by smooth, glistening, pink epithelium Normally, none of the reddish-pink, bright endocervical mucous membrane is seen, but occasionally a symmetrical area of it is exposed, extending peripherally from the os for a distance of 1–2 cm A congenital defect, this indicates lack of normal development, other manifestations such as infantile or juvenile uterus should be looked for It is frequently associated with dysmenorrhea and endometriosis Exposed endocervical epithelium of congenital origin is distinguished from that due to inflammation or tumor by its symmetrical distribution around the os.

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Leukoplakia. *Common on the cervix, especially where the two epithelia meet,* leukoplakia is represented by small, dull white, slightly raised areas. It may reflect thickening of the horny layer of epithelium similar to keratosis on the skin, or growth of atypical cells involving all the layers of epithelium which *represent the earliest form of cervical carcinoma* (*cancer in situ*) The two types can be differentated only by microscopic examination. Invisible areas of leukoplakia can be brought out and obvious areas made more prominent by staining the cervix with Schiller's or a slightly stronger iodine solution. Leukoplakia *and carcinoma do not take the stain but retain their original color;* normal endocervical epithelium stains light brown, normal cervical epithelium, dark brown *In any case of leukoplakia, biopsy is essential to exclude carcinoma*

Carcinoma. This is the most common malignant tumor of the female genitals *It is always to be suspected under the following circumstances.*

1. Bleeding, even of slight degree, between periods, after menopause, or following coitus or douching

2. Appearance of vaginal discharge or increase of previously existing dis*charge especially if it is irritating, foul, watery, or bloody*

In its earliest stage (*cancer in situ*), carcinoma of the cervix cannot be clinically differentiated from other forms of leukoplakia A biopsy should be made of any suspicious area, although examination of vaginal secretion by the Papanicolaou *method will often establish the diagnosis. (This test should be performed not* only as part of a routine pelvic examination but especially whenever the possibility of intrapelvic malignant disease is entertained.)

Later the tumor appears as a red, raised, granular, usually bleeding lesion *at the junction of the two epithelia The surface has a sand-paper feel, in con*trast to the soft, velvety sensation imparted to the palpating finger by normal mucosa A carcinomatous lesion can be readily penetrated by a blunt probe whereas resistance is encountered in normal epithelium (*Chrobak test*)

With progression, the cervix becomes hard, nodular, and irregular; friable nodules which break off to leave bleeding points may be evident, although occasionally the tumor extends beneath the surface, whereupon bleeding is not a feature

In the far-advanced case, the organ is large, hard, fixed, and almost always bleeds when touched Invasion of adjacent structures may be indicated by visible extension of tumor onto the vaginal wall or, on rectal palpation, by thickening of ureterocervical segment, or thickening and fixation of the broad ligaments Large or small hemorrhages characterize this stage Sometimes carcinoma grows inside the cervical canal, spreads upward and outward toward the periphery and, because of failure to present early visible or palpable signs, is overlooked until far advanced. Here the first indication is likely to be a disturbance of micturition based on involvement of bladder floor Pain occurs only when the tumor has invaded an adjacent structure such as *the bladder, rectum, or a pelvic* nerve; it is usually felt low in the pelvis or back, or in the thighs Ureteral obstruction with resultant hydronephrosis is not uncommon; it reflects direct invasion of the bladder or pressure from enlargement of nearby nodes. The most



Fig 33 22 Procidentia of cervix with extensive carcinoma (Courtesy Pondville Hospital, Massachusetts Dept of Public Health)

likely metastatic spread is to the lungs and supraclavicular nodes via the periaortic chain, and to bones

Body of Uterus

Malpositions. As noted earlier, the normal uterus is anteverted, tending to be horizontal and slightly anteflexed The following variants of position are commonly encountered

Anteflexion Usually an indication of lack of normal development and found with a small uterus, this is marked by a sharp angulation palpable between the anterior wall of the cervix and the fundus

Retroversion The body is turned backward When moderately displaced, it cannot be felt, in pronounced (third degree) retroversion, it is felt behind the cervix through the vagina or rectum, but not with the abdominal hand. The cervix points downward or anteriorly, depending upon the degree to which the fundus is displaced

Retrocession The uterus has sagged backward so that it occupies a position further posteriorly in the pelvis than normal, that is, it lies nearer the sacrum. The cervix is directed in the long axis of the vagina or slightly forward.

Retroflexion Usually occurring in association with retroversion, this is marked by an angle palpable posteriorly between the cervix and body

Prolapse Relaxation, stretching, and perhaps tearing of its supporting structures incident to childbirth permit the uterus to sink downward in the pelvis The cervix is low and lies in the vaginal axis, both it and the fundus are often enlarged owing to congestion from impaired circulation. Perineal lacera-

tion, rectocele, and cystocele are usually present Prolapse (or even procidentia) is sometimes encountered in the *nulliparous*.

PROCIDENTIA This is pronounced prolapse with the cervix presenting at or outside the vaginal introitus. *In addition to the associated variants* just mentioned, one may also find cervical leukoplakia or ulceration, especially in the long-standing case.

Enlarged Uterus. Common causes are:

1. Pregnancy. The uterus is *smooth, rounded, symmetrical*, and soft.

2 Subinvolution following *pregnancy Here one finds symmetrical enlargement*, usually with *retroversion*.

3 Liomyoma (fibroid tumor). The organ is symmetrically irregularly enlarged, soft or firm, *pedunculated growths extending in any direction may be* felt

4. Endometrioma *Irregularity, enlargement, and firmness are the rule*

5 Endometrial carcinoma. One may find no enlargement, or a moderate degree of symmetrical enlargement.

6 Pyometria. The uterus is large, soft, and tender.

Small Uterus. Before menopause, *small uterus is usually a reflection of under*development, anteflexion is also present. Following menopause, the organ gradually becomes smaller and ultimately so shrunken that it is barely felt, it is often moderately retroverted

Liomyoma. Excessive bleeding at catamenia, prolonger catamenia, or continuous bleeding is the most important sign of fibroid tumor In most cases it is due to endometrial hyperplasia created by a coexistent ovarian dysfunction but occasionally to distortion of endometrial cavity and pressure on opposite endometrial surface by a submucous tumor. Pain is rare, occurring only from pressure on the bowel, rectum, or bladder, or from attempts of the uterus to expel tumor presenting in its cavity Vaginal discharge, usually foul and watery, sometimes blood-stained, is common as a result of infection of a submucous growth or a pedunculated tumor in the canal Anemia, secondary to prolonged bleeding, is common and may create the symptoms which first bring the patient to her physician Pelvic examination reveals a symmetrical or asymmetrical, enlarged uterus firmer than the pregnant uterus, or one or more hard, circumscribed tumor masses in the lower abdomen Submucous fibroid, often the cause of severe bleeding, cannot be felt *Fibroid is often difficult to distinguish from a pregnant uterus or an ovarian tumor or cyst*

Endometriosis. One or more ectopic growths of endometrium develop *in the* pelvis or abdomen They are most common in the uterus and cervix (*adenomyomata*), and ovary but may also be *found in the rectum, sigmoid, Fallopian* tubes, appendix or lower ileum, less often in the umbilicus, inguinal canal, bladder or vulva *but rarely above the level of the umbilicus* Growths have been reported in the pleural cavity and muscle of thigh and arm Acting like endometrium, *the tumor tissue is influenced* by ovarian hormones and periodically either menstruates or undergoes changes comparable to menstruating endo*metrium; monthly pain*, often wrongly attributed to dysmenorrhea, is created

by swelling and pull on adhesions. Endometriosis is especially to be suspected if the pain begins a day or so before and lasts throughout the period Depending on the site of the lesions, the discomfort may be located by the patient in her lower back or abdomen, rectum, or bladder Uterine adenomyoma increases menstrual bleeding. *During pregnancy, lactation, and following menopause, symptoms are absent.*

Local examination is often negative If the uterus is involved, it may be somewhat enlarged and irregular Endometriosis in the pelvis is usually accompanied by fixation of the uterus and adnexa and, if in the posterior cul-de-sac, can often be positively identified by feeling, on rectal or vaginal examination, small, rough, shotty, or beaded areas behind the cervix in either uterosacral ligament An ovarian lesion may produce a palpable mass in a vault. The umbilicus, if involved, becomes blue during menstruation

Progressive intestinal obstruction is occasionally caused by invasion of the rectum, sigmoid, or, rarely, the lower ileum, hydronephrosis, by pressure of tumor on a ureter

Carcinoma of Body of Uterus. Usually developing after menopause, in contrast to cancer of the cervix which occurs in any age group, this is marked by irregular bleeding or staining, although occasionally a watery or blood-stained discharge will appear first If the disease develops before menopause, *profuse and prolonged menstrual bleeding may antedate intermenstrual variants.* When the tumor is well-established, pain similar to that of catamenia may occur; it reflects efforts of the uterus to expel the mass Examination can be entirely negative or show a symmetrically enlarged uterus Sometimes metastatic growth in the ovaries will make them palpable or metastatic nodules will be found in the cervix or vagina The Papanicolaou test may establish diagnosis, if *it is negative, curettage or biopsy is indicated Local invasion and distant metastasis follow the same patterns as cervical carcinoma*

Ovaries and Fallopian Tubes

Bleeding Graafian Follicle. Especially in an adolescent or young woman, an episode of low abdominal pain which suggests appendicitis and occurs from approximately 14–10 days prior to an expected menstrual period, may well be due to bleeding from the follicle at the site of ovulation The pain, experienced on either side or in midline, begins very abruptly and is usually more intense than that of early appendicitis, *overlying tenderness and perhaps spasm are present As a rule, local tenderness can also be elicited in a vault or the posterior* cul-de-sac Slight uterine bleeding may occur but not often enough for it to be dependable as a diagnostic criterion Unless the picture is clearly one of appendicitis, observation instead of immediate operation is justifiable, provided there are no untoward developments. In contrast to appendicitis, the symptoms usually disappear within 24–48 hours Occasionally the bleeding will persist and, if profuse, create the picture of diffuse peritoneal irritation, or signs reflecting serious blood loss In the doubtful case, the presence of intraperitoneal bleeding can be established by placing the patient in the Trendelen-

burg position· subphrenic peritoneal irritation due to the gravitated blood will produce pain in one or both shoulders

Sometimes a comparable episode will occur during the latter part of the intermenstrual interval owing to bleeding from an imperfect corpus luteum or stretching of an abnormal corpus hemorrhagicum.

Tubal Pregnancy. This should be suspected when a woman of childbearing age suddenly develops abnormal vaginal bleeding or staining, especially when accompanied by low abdominal pain As a rule, one or two periods will have been skipped Breast changes may have begun. Symmetrical enlargement of the uterus suggestive of early pregnancy is usually found, movement of the fundus and adnexa produced by upward pressure on the cervix causes acute pain. Tenderness high in one vault can often be elicited The enlarged tube may or may not be palpable. Prior to abortion or rupture, tubal pregnancy can rarely be diagnosed with certainty on the evidence presented. If it is suspected, further investigations are essential. Demonstration of blood in the posterior cul-de-sac by needle or incisional colpotomy is regarded as virtually pathognomonic. Some surgeons use the culdoscope in an effort to visualize the affected tube. Others by-pass these procedures, since they regard the picture *just described as a definite indication for surgical interference*

TUBAL ABORTION OR RUPTURE. This is marked by abrupt appearance of severe unilateral lower abdominal pain, tenderness and spasm, and faintness or indications of peripheral circulatory collapse based on intraperitoneal hemorrhage The insult is usually accompanied by vaginal bleeding which may be less than or equal to normal menstrual flow, the blood comes not from the ruptured tube but from the uterine mucosa which is no longer supported by placental endocrine activity and the ovarian hormones which depend on the latter Leukocytosis is the rule Pelvic or rectal examination will disclose a tender mass in the homolateral vault and, occasionally, tenderness and fulness in the posterior cul-de-sac reflecting a collection of blood. Motion transmitted to uterus and adnexa by pushing on the cervix creates exquisite local pain Blueness of the umbilicus (*Cullen's sign*) is occasionally seen as a result of intraperitoneal bleeding In the doubtful case, presence of free blood in the peritoneal cavity may be established by placing the patient in the Trendelenburg position (*see above*). *Some patients give no history of missed periods nor show, at the time of the episode, evidence of abnormal bleeding from the uterus or into the abdomen*

Rarely in tubal pregnancy, bleeding into the tube itself without immediate rupture or abortion, causes the fetus to die *in situ* Here the picture is one of transitory right or left lower quadrant pain, with vaginal bleeding which may persist for several days or longer Unilateral tenderness and a palpable mass are the rule; evidence of gross intraperitoneal hemorrhage is lacking

Acute Gonorrheal Salpingitis. Failure to obtain a history of gonorrheal infection or sexual exposure does not exclude the disease. Bilateral—rarely, unilateral—lower abdominal pain, occasionally accompanied by nausea and vomiting, is the outstanding symptom. Temperature and leukocyte count are elevated Abdominal tenderness and spasm are found, usually directly over the pubes.

The cervix is red, edematous, and exudes a mucopurulent discharge in which gonococci may be found within the first few weeks but, because of contamination, are rarely detectable later. Evidence of infection of Skene's or Bartholin's glands is common. Any attempt to move the uterus by pressure on the cervix provokes exquisite pain. Palpation of the lateral vaults reveals tenderness and often a large or small mass on one or both sides Occasionally the tenderness is directly behind the cervix. On bimanual palpation, the intravaginal finger may feel nothing abnormal, but a mass will be discovered by deep pressure with the abdominal hand. Rectal palpation is important because the finger is not restricted, as it is in the vagina, by the fold of vaginal wall behind the cervix; it reaches higher, and thus is more likely to detect tenderness or a mass in the posterior cul-de-sac or a broad ligament

Differentiation between acute salpingitis and acute appendicitis is often difficult In salpingitis, the trouble is more likely to begin immediately after catamenia, pain and tenderness are lower and less apt to be limited to the right side. The woman with salpingitis appears more ill than one with appendicitis who has local signs of equal degree Her leukocyte count is higher—18,000–20,000 or more—a figure not ordinarily reached in appendicitis except in the presence of abscess or general peritonitis

In recent years, because of the widespread use of antibiotics, the picture of acute gonorrheal salpingitis is rarely as clear-cut as described

Puerperal Infection. This follows abortion, miscarriage, or labor Currently, thanks to antibiotics and improved obstetrical methods, it is much less common than heretofore Since most cases are due to *streptococcus* they are first characterized by cellulitis of the uterus and parametrial tissues Chills, high fever, and other signs of severe sepsis are present The uterus is tender and perhaps enlarged, the broad ligaments tender and thickened Occasionally one can feel a mass representing pelvic or broad ligament abscess General peritonitis, thrombophlebitis of the pelvic veins, and septicemia are possible but rare complications

Chronic Salpingo-oophoritis This results from continuation of gonorrheal or puerperal infection, the initial attack of which may or may not have been recognized Symptoms are low abdominal or pelvic pain, pressure, bearing-down or heaviness, almost always worse at catamenia Menorrhagia or other menstrual disturbance, and leukorrhea are often but not necessarily present. In the mild case, absence of local signs may make diagnosis impossible When the process is more extensive, one will find evidence of cervicitis and thickening of the broad ligaments. In gonorrheal infection, a cystic or hard mass representing a collection of fluid or pus within the tube can often be felt uni- or bilaterally (*hydro- or pyosalpinx*)

The diagnosis of chronic salpingitis may be confirmed by x-ray examination of the tubes following injection of an opaque medium; a non-patent tube usually means an inflammatory process This test should not be performed unless acute disease has been excluded

Tuberculous Salpingitis. This may take the form of endosalpingitis with

slight-to-great enlargement of the tubes easily felt bimanually, or a perisalpingitis which usually accompanies tuberculous peritonitis and *cannot always be detected on physical examination. Except when the history points to gonorrhea or other acute infection as the cause of a chronic pelvic process, tuberculosis cannot be excluded on clinical grounds, it must be strongly suspected if this disease is found in a lung, the urinary tract, or elsewhere.*

Pelvic Peritonitis. ACUTE. This occurs as a complication of acute salpingitis, puerperal infection, pelvic appendicitis, or other intra-abdominal inflammatory process. The entire pelvis is acutely inflamed Lower abdominal and intrapelvic pain and tenderness, and moderate to severe systemic response are present Exquisite pain can be elicited by pressing the cervix upward Downward bulging or a palpable mass in the posterior cul-de-sac or a vault indicates pelvic cellulitis or a collection of pus (*pelvic abscess*).

CHRONIC The end-result of an acute process, chronic peritonitis is characterized by adhesions which, as they contract, cause displacement, distortion, and consequent interference with function of pelvic organs

The term *pelvic inflammatory disease* theoretically refers to any of the above infections; in common usage, it is accepted as indicating gonorrhea unless otherwise specified In the chronic case, the nature of the infection can often be determined only by operation Any such process is likely to be responsible for infertility

Ovarian Tumor. The varieties are so numerous that a classification is outside the scope of this book Ovarian tumor can occur at any time—before, during, or after menstrual years Depending on its histogenesis, it can be large or small, solid or cystic, benign or malignant, functioning or non-functioning Primary tumor must be excluded in the presence of a menstrual deviation not attributable to some obvious cause, and in any female showing precocious puberty or a trend toward masculinization However, these variants do not necessarily indicate ovarian disease, they may be a reflection of an adrenal or pituitary disorder with or without ovarian functional participation Occasionally an asymptomatic tumor is found on routine examination, or during a search for the cause of sterility. Metastatic disease is not uncommon, especially from the stomach (*Krukenberg tumor*), other segment of the gastro-intestinal tract, breast, or uterine fundus.

Local manifestations, as a rule, are dependent on size A small neoplasm may be symptomless and impalpable or barely palpable In the absence of any systemic change or regional discomfort, it may be overlooked for a long time, perhaps becoming manifest only when signs related to peritoneal extension or distant metastasis appear.

A large tumor causes pressure symptoms bearing-down sensation, backache, frequency or other urinary disturbance, or increasing constipation Here one may find a definable, usually somewhat movable mass of any size, or the tumor may be so huge that it virtually fills the abdomen and gives it a swollen, rounded appearance similar to that caused by ascites Low-grade fever due to necrosis

incident to rapid growth is sometimes encountered If the pedicle becomes twisted, a picture resembling acute appendicitis or other intra-abdominal insult such as intestinal perforation or obstruction will develop. A large cyst or tumor is most easily mistaken for pregnancy, uterine fibroid, or ascites If, on pelvic palpation, the uterus can be identified, and a mass felt in one or both vaults, ovarian tumor is most likely, but one may be misled by a subserous fibroid tumor attached to the uterus by a pedicle The differences on physical examination between large ovarian cyst and ascites have already been discussed (*see* Chap. 29). Demonstration of fluid does not necessarily exclude benign or malignant ovarian tumor, either of which can cause ascites; sometimes by ballottement over the lower abdomen, a tumor can be felt beneath the fluid but failure to detect it does not rule it out

In malignant disease, extension to the opposite ovary, uterine fundus, abdominal lymphnodes and peritoneum is the rule, eventually distant metastases will develop, especially in the liver, lungs, and long bones.

Frequently fibroma of the ovary, irrespective of its size, will be accompanied by benign ascites and hydrothorax (*Meigs' syndrome*) The fluid will disappear following removal of the tumor. Intrathoracic fluid can also appear in other forms of benign or malignant neoplasm, although in the malignant case, it usually represents pleural metastasis, it is, on rare occasions, benign

Pregnancy

Early Pregnancy. The first symptoms, which usually appear from the sixth to eighth week following conception, are lassitude, irritability, depression, anorexia, nausea and vomiting, especially in the morning, tingling of the breasts, and frequency of micturition Loss of weight is common within the first 3 months, even in the absence of anorexia or vomiting The first signs, always more easily demonstrable in the first than in subsequent pregnancies, appear at about the same time firmness of breasts with enlargement of Montgomery's glands, slight blueing of vaginal mucosa, perhaps increased vaginal secretion, and softening of the cervix From the nipples, a small amount of straw-colored secretion (*colostrum*) can be expressed If the uterus is anterior, slight enlargement and a softer than normal consistency can be made out bimanually The Aschheim-Zondek or other test for chorionic gonadotrophic hormone will usually be positive by the third week after conception, and if so, is all but pathognomonic

In the second or subsequent pregnancies, palpation of the cervix and fundus is of little value until 8–10 weeks after conception, and until then diagnosis cannot be positively established without a biologic test. In a fat or muscular woman or one with retroversion, it is difficult to palpate the fundus satisfactorily within the first 3 months

Mid-pregnancy. After the third month, lassitude and digestive disturbances are usually at an end, the disposition is brighter, and weight has begun to increase Enlargement of Montgomery's glands is greater, the areolae and nipples begin to show pigmentation, and vaginal secretion and blueing are more marked. The uterus is now obviously enlarged and, except in the obese, readily

felt by abdominal palpation, its upper border averaging 10–12 cm. above the pubis. During the fifth month, lower abdominal swelling becomes apparent; in a thin patient the experienced observer may be able, by abdominal palpation, to identify the hard fetal head and firm resistant back. The mother can be aware of fetal movements although they may not appear until later. With the fetoscope—an ordinary stethoscope equipped with a large Bowles type receiver—one may hear, usually near the umbilicus, the tic-tac sounds of the fetal heart. Occasionally as early as the sixth month, placental souffle is audible *(see below)*.

Late Pregnancy. By the seventh month enlargement of the abdomen is pronounced. The added weight forces the mother to stand on a wide base with shoulders thrown back, and to walk in a waddling fashion. She may complain of dyspnea accentuated by effort, and palpation or consciousness of forceful heart beat. The areolae and nipples are darker, the latter enlarged. A narrow line of pigmentation extending upward in midline from the pubis to above the umbilicus and shading off toward the ensiform tip, has appeared *(linea nigra)*. The first heart sound may be increased at the apex, a systolic murmur at the pulmonic area or apex is likely. The ankles may be somewhat edematous. The *uterus extends 26–28 cm. above the symphysis*. The hard, rounded, well-circumscribed fetal head, the somewhat softer, larger, and less well-defined back and buttocks, and one or more limbs can usually be identified. In most cases, the fetus lies with the head toward the symphysis and the back higher up facing anteriorly, posteriorly or laterally. With the fetoscope, the fetal heart can now be heard everywhere over the abdomen, usually loudest just above or below the umbilicus. Especially if the placenta is anterior, a swishing sound synchronous with the mother's systole is also heard near the umbilicus *(placental souffle)*. In the eighth and ninth months the findings are essentially the same but the fetus is larger.

The blood may show diminished red cells, hemoglobin and hematocrit, slight leukocytosis and some elevation of sedimentation rate.

Abortion. Expulsion of the ovum or fetus from conception to the time of viability, usually the 28th week, is called *abortion*, from then until term, *premature labor*. In some clinics, the word *miscarriage* is applied to loss of the fetus during the middle 3 months, in others, it is used interchangeably with both abortion and premature labor. The important indications of abortion are vaginal bleeding and lower abdominal cramps but it must be remembered that if these occur early in pregnancy they may be due to tubal abortion or rupture. In the late months, the same indications can herald premature delivery but are more likely to be a reflection of placenta praevia or separated placenta.

Hydatidiform Mole. Owing to changes secondary to degeneration of blood vessels in the stroma of immature chorionic villi, the villi are converted from minute t[illegible]
process b[illegible]
months. [illegible]
enlargement greater than is consistent with the gestational age. In many cases, one finds associated albuminuria and hypertension. Provided uterine enlarge-

ment from fibroid tumor or multiple regnancy can be excluded, failure to identify fetal bones on carefully taken x-ray films after the uterus has reached the size of 4 months' pregnancy, is highly significant As a rule, the levels of chorionic gonadotrophic hormone in blood and urine are much higher than in normal pregnancy. Passage by vagina of clumps of the typical vesicles is pathognomonic, but unfortunately this seldom occurs The bleeding may lead to a mistaken diagnosis of abortion

Chorionepithelioma. This is a *rare malignant tumor of trophoblastic origin* About one-half the cases are preceded by hydatidiform mole, about one-quarter follow abortion, and the remainder develop following normal or ectopic pregnancy Persistent bleeding, *or bleeding recurring weeks or months following* normal delivery, abortion or hydatidiform mole, should alert one to the possibility of this misfortune The uterus is of *normal size, or only slightly enlarged.* Because the tumor is highly malignant and metastasizes early, a secondary growth especially in a lung or the vagina may be the first indication of trouble As in hydatidiform mole, the chorionic gonadotrophic hormone in the blood and urine is greatly in excess of that found in pregnancy

34

THE PELVIC GIRDLE AND LOWER EXTREMITIES

THE PELVIC GIRDLE

Strain or Separation of Symphysis Pubis. Occurring during labor or from violent injury, either is marked by regional pain and tenderness In the female, the latter can best be detected by vaginal palpation It is often accompanied by sacroiliac strain Separation is confirmed by x-ray

Strain of Sacro-iliac Joint (*see* Chap. 7)

Fracture of Pelvic Girdle. Extreme violence, particularly a crushing accident, may cause fracture at any point in the pelvic ring The symptoms, varying with the degree of displacement, are chiefly extreme pain and tenderness over the site of injury although sometimes it is difficult for the patient to pinpoint the former Pain is intensified by any movement or by manual compression of the iliac crests Shock is often pronounced Severe fracture may also cause injury to the pelvic nerves or viscera, especially vesical or urethral rupture

Fracture of Acetabulum. Resulting from a direct blow on the trochanter, this produces local pain, limitation of hip motion, and shortening of the extremity. Diagnosis is confirmed by x-ray

Coccygodynia. Pain in the region of the coccyx, usually aggravated by sitting, is most commonly due to a direct blow such as a fall on the buttocks but also occurs following parturition, and sometimes without known cause Tenderness of the coccyx or the sacrococcygeal junction can be elicited by external or rectal palpation, intense pain by local manipulation In some cases, the coccyx is tipped anteriorly or deviated laterally and is more freely movable than normally The pain may persist for months or years

Bursitis. The bursa overlying the ischium is often inflamed in equestrians and persons whose occupations require long periods of sitting, especially if constant motion of the lower extremities adds local friction (*weaver's bottom*) Pain, tenderness, and sometimes swelling over the ischial tuberosity are present Regional tenderness, and pain on motion of the thigh, especially flexion and inward rotation, suggest inflammation of the bursa over the greater trochanter.

THE GROINS

Enlarged Lymphnodes. For practical purposes, the nodes in each groin can be grouped as follows: (1) *inguinal*, situated in the subcutaneous tissue about

Poupart's ligament and receiving drainage from the external genital and perineal regions, lower abdomen and back, buttock and upper part of the thigh; (2) *saphenous*, situated about the saphenous opening and receiving drainage from the lower thigh, leg, and foot Either or both groups may become involved in mononucleosis, leukemia, lymphoma, and other diseases causing generalized lymphnodopathy.

Acute or chronic inflammation of a pertinent structure causes *enlargement of the inguinal group* Suppuration and sinus formation usually mean chancroid, tuberculosis or lymphogranuloma venereum Hard, shotty enlargement is likely in cancer of the anus, penis, scrotum, vulva, or lower vagina From the other genital organs, lymphatic spread of a malignant process is to the nodes within the pelvis or abdomen; involvement of the inguinal group occurs, if at all, only as a late manifestation and is then a reflection of extension to its drainage area as, for example, invasion of the scrotal wall *in carcinoma of the testicle*

Enlargement of the saphenous nodes reflects trouble in the lower extremity, except *for the upper thigh*

Hernia (*see* Chap 29)

Encysted Hydrocele of Spermatic Cord. This is felt in the inguinal canal as a circumscribed, elastic swelling which gives an impulse on cough, is dull on percussion, and not completely reducible *If the cord is pulled downward, the swelling moves with it* In the female, a patulous process of peritoneum sometimes accompanies the round ligament partway through the inguinal canal, if it becomes constricted, the portion beyond the obstruction may, by secreting fluid, create a cystic swelling analogous to that just described (*hydrocele of canal of Nuck*)

Cryptorchism (*see* Chap 33)

Psoas Abscess. If this points in the inguinal region it appears as a rounded, fluctuant, usually painless swelling below Poupart's ligament, usually lateral to the femoral vessels

Aneurysm of External Iliac Artery. Not common, this produces expansile pulsation in the inguinal region and reduction of the corresponding femoral pulse A bruit may be heard

Filarial Lymphangiectasis. Soft to tense, lobulated, usually non-tender, swellings in the groin may be found in filariasis as a result of obstruction and dilatation of the lymphatic vessels

A swelling in the groin should never be incised until aneurysm and hernia have been excluded

THIGHS, LEGS, AND FEET

Position and Deformities

Abnormal position and deformities of a lower extremity result from a variety of causes, chiefly bone and joint diseases such as rickets, arthritis, tuberculosis, Paget's disease, improperly reduced fracture or dislocation, and atrophy, paralysis or contracture of one or more muscle groups Associated with some of these

are found disturbances of gait, the most striking of which are described elsewhere (*see* Chap 3 and 36)

Shortening. When not enough to be obvious, inequality of leg length can be detected by careful measurements. On each side, the distance from the anterior superior spine of the ilium to the internal malleolus is determined The patient must be supine on a table or flat bed with his lower extremities completely extended, his feet equidistant from the projection of midline of the body, and his anterior superior spines in the same horizontal line False readings will be obtained if the pelvis is tipped (indicated by one anterior superior spine being higher than the other) or the feet not equidistant from midline. Shortening results from birth injury, dislocation of hip, improperly reduced fracture of a long bone, and following poliomyelitis, tuberculosis, rachitis, osteomyelitis, or other disturbance affecting bone growth

Genu Varum. Usually the result of rickets in childhood, bowleg is characterized by lateral curving of the tibia perhaps accompanied by anterior curving, and, in the severe case, similar changes in the femur Bowing also occurs in achondroplasia, Paget's disease, and following improperly reduced fracture

Genu Valgum. Knock-knee is usually congenital or a sequela of rachitis The deformity may be apparent only when the patient is standing The normal inward inclination of the femur from above downward is exaggerated, bringing the knees together and resulting in outward inclination of the lower legs which prevents close approximation of the feet

Pes Planus. Flat foot may be congenital or acquired, and the result of an osseous, ligamentous or muscular variant Contributing factors are excessive use following a period of disuse, long periods of standing, faulty footwear, improper walking, weakened musculature from any cause, and obesity The metatarsal and longitudinal arches show pronounced relaxation or flattening, more marked when the weight is thrown on the affected foot. The latter is in the position of pronation and, in the long-standing case, shows diminished flexibility It may be symptomless or give the picture of foot strain

Pes Cavus. Contracted foot may be congenital, or acquired from habitual use of high heels, poliomyelitis, local neuropathy, or other cause of muscular contracture The longitudinal arch is abnormally high, while the toes tend to be dorsiflexed at the metatarsophalangeal joints

Talipes Equinovarus. Clubfoot is a congenital deformity marked by equinus (downward pointing), varus (inversion) and adduction The patient walks on the lateral aspect of the foot with resultant functional discomfort and development of corns, calluses, and inflamed bony prominences.

Hammer Toe. Usually congenital, this is a flexion-contracture of one toe, almost always the second. Pain may occur at abnormal pressure points

Saber-shin. Due to periosteitis, forward curving of the anterior border of the tibia, sometimes with nodular thickening, is not uncommon in congenital and late acquired syphilis

Enlarged Epiphysis. A frequent manifestation of rachitis, this is usually encountered in the lower extremity at the lower end of the tibia and fibula.

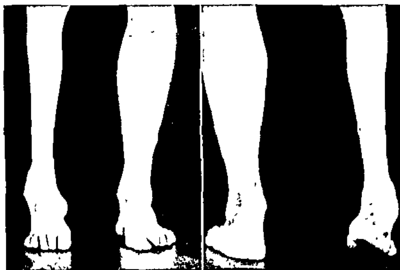


FIG 34 1 Equinovarus deformity with high longitudinal arch of foot and atrophy of leg muscles due to anterior poliomyelitis

Hallux Valgus. Congenital, or acquired through prolonged use of faulty footwear, this deformity is characterized by medial displacement of the head of the first metatarsal bone, causing a prominent swelling of the inner aspect of the foot at this point (*bunion*) and lateral bending of the great toe sometimes so extreme that it overlaps the second It is asymptomatic unless the bursa overlying the joint becomes irritated or inflamed by pressure or friction, whereupon severe pain, redness, swelling, and tenderness will appear. The severe case may be mistaken for gout



FIG 34 2 Congenital club feet

Atrophy

Atrophy of *a part or all* of *one* extremity may occur in anterior poliomyelitis, disease of the hip or knee, hemiplegia, certain less common neurologic disorders, and following *prolonged disuse* from any cause. Pronounced atrophy is most often due to destruction of the anterior horn cells or permanent injury of a peripheral nerve

Atrophy of part or all of *both* extremities is most likely due to multiple neuropathy, anterior poliomyelitis, the Charcot-Marie-Tooth *type of atrophy*, progressive muscular atrophy, Friedreich's ataxia, transverse myelitis, or bilateral bone or joint disease

Abnormalities of Motion

Tremors, spasms and choreiform movements of the lower extremities are due to the same causes which produce these variants in the arms and hands

Paralysis

Flaccid Paralysis. One or both lower extremities may be partially or totally paralyzed in poliomyelitis In peripheral neuropathy, weakness or paralysis is uni- or bilateral, depending on the cause, when the neuropathy is due to alcohol, dietary deficiency, lead or other toxic agent, foot-drop is likely As a rule, uni- or bilateral flaccid paralysis promptly follows cerebral thrombosis, embolism or hemorrhage, and severe spinal cord injury, depending on the nature and severity of the insult, flaccidity may persist or be followed by spastic paralysis Among the other causes of bilateral flaccid paralysis or weakness are progressive muscular atrophy, cauda equina tumor, and the Charcot-Marie-Tooth type of atrophy

Spastic Paralysis. Unilateral spastic paralysis most commonly occurs with cerebral vascular insult, tumor, syphilis or injury The muscles of the arm and face on the same side are similarly affected except when the fibers from the leg area alone are involved, as might happen, for example, in brain tumor, abscess, or small infarct Bilateral spastic paralysis usually means spinal cord disease, most commonly multiple sclerosis, subacute combined degeneration or transverse myelitis due to infection, injury involving the cord, or cord compression from intra- or extramedullary tumor. It is also seen in some birth injuries to the brain, in Friedreich's ataxia, and certain rarer disorders.

Hysteria may simulate any of the common forms of paralysis but is betrayed by the fact that reflexes are not usually abnormal, and motor and sensory changes do not conform to known patterns of neurologic disintegration

Injuries

Fracture of Hip. Fracture of the neck of the femur is common in the aged usually following a fall, or perhaps minor trauma such as a stumble or misstep;

it may be that the break occurs spontaneously and the fall is the result, not the cause. In a child or young adult, fracture here occurs only with severe direct or indirect trauma. Local pain, tenderness, and perhaps discoloration are present, pain on motion is the rule The patient may be unable to walk or stand. Marked external rotation and perhaps shortening of the extremity are found If the fracture is impacted, these two changes are minimal or absent, and walking may be possible with only moderate pain.

Dislocation of Hip. ACQUIRED DISLOCATION Resulting from violent trauma, usually a twisting injury, this is most often encountered in young adults Local pain is present The signs are inability to stand or walk, restriction of motion, regional tenderness, shortening, and deformity, the character of which depends on the location of the femoral head with respect to the acetabulum

CONGENITAL DISLOCATION Uni- or bilateral, this is first noted when the child begins to walk. If unilateral, a characteristic limp is noted, if bilateral, a waddling gait The femoral head is easily palpable, usually posterior to and above the acetabulum, and is abnormally mobile, being easily pushed up and down (*piston mobility*). Bilateral dislocation is characterized also by posterior prominence of the trochanters and exaggeration of the lumbosacral curve, giving a sway-back appearance In either type, *Trendelenburg's sign* is positive if the child stands with his weight thrown on a dislocated hip and flexes the thigh and leg on the other side, the pelvis will tip *downward* toward the flexed, non-weight-bearing side, whereas in one standing on a normal extremity, with his other leg raised, the pelvis will tip *upward* toward the non-weight-bearing side

Fracture of Femoral Shaft. Occurring in the upper, middle, or lower third, this creates pain, tenderness, swelling, shortening, deformity, and abnormal mobility at the site of the break In a child, separation of the upper femoral epiphysis can occur with or without trauma, in the latter instance, it is thought to be related to some endocrine disturbance Pain and tenderness, limitation of hip motion, especially flexion and rotation, and possibly shortening are characteristic Separation of the lower femoral epiphysis, always due to severe trauma, gives the usual signs of fracture

Sprain of Knee. Sprain of the internal lateral ligament due to sudden abduction of the leg on the thigh is the commonest injury to the knee joint Pain, swelling, and tenderness, usually localized to the femoral attachment of the ligament, are typical

Injury to Semilunar Cartilage. Resulting from sudden wrench or twist with the knee flexed, this causes severe pain, swelling, and tenderness along the joint line medially or laterally Inability to extend the leg on the thigh (*locking*) may occur, it can usually be relieved by active or passive manipulation Recurrences are common even after mild trauma

Fracture of Knee. Sudden contraction of the quadriceps muscle with pulling apart of the patella is a common injury Severe pain, swelling, inability actively to extend the leg on the thigh, and a palpable transverse defect at the site of the fracture are the characteristic manifestations Fracture of the lateral head of

the tibia (*bumper fracture*), caused by a blow on the outer aspect of the knee, and fracture of the tibial spines are not infrequent.

Loose Bodies in Knee. Loose bodies in a joint (*joint mice*) may appear in adolescence as a result of local nutritional disturbance, and can also occur following trauma and in association with tuberculosis, syphilis, degenerative arthritis and certain other less common disorders In the knee they may be asymptomatic or create some degree of continuous or intermittent pain, swelling, and limp. Aggravation by trauma or excessive use is the rule; episodes of locking are likely. If they become calcified the bodies can be seen by x-ray

Fracture of Leg. Either the tibia, fibula, or both may be broken in their upper, middle, or lower thirds Fracture of the lower end of the tibia and fibula (*broken ankle*) is common as a result of a stumble, slip on a curb or loose stone, or a fall with the weight of the body thrown on the foot while it is twisted The foot is usually everted or inverted When the trauma is less severe the lower end of the fibula is fractured and the internal malleolus pulled off or the internal lateral ligament badly torn (*Pott's fracture*). Ankle fractures are often accompanied by dislocation, usually posterior, of the foot on the leg, giving marked deformity, shortening of the foot, and pronounced regional swelling

Sprain of Ankle. Trauma, similar to but less severe than that causing ankle fracture, creates tear of the external lateral ligament or, less often the internal ligament Local pain, tenderness, swelling, and usually discoloration occur Injury to the anterior tibio-fibular ligament is marked by local pain, swelling, and tenderness anteriorly between the lower ends of the tibia and fibula

In severe sprain a small chip may be pulled loose from a malleolus or one of the tarsal bones (*sprain fracture*)

Rupture of Tendon. Due to sudden acute strain, rupture of the *plantaris* tendon causes sharp, abrupt pain in the posterior aspect of the lower leg increased by walking, passive dorsiflexion, and active plantar-flexion of the foot Tenderness, swelling, and ecchymosis occur in mid-calf

A much rarer injury, rupture of the *Achilles* tendon results from violent trauma, especially a direct blow or abrupt stress forcing the foot into dorsiflexion Sudden sharp pain is felt in the lower part of the leg posteriorly, it is aggravated by passive dorsiflexion but this movement may show greater range than on the uninjured side. Active plantar-flexion is impossible Spot tenderness is present over the tendon, a transverse defect at the site of rupture may be detectable Edema and ecchymosis of the ankle and foot develop promptly The former is likely to prevent palpation of the transverse defect

Foot Strain. Acute or chronic foot strain can be produced by improper or an unaccustomed amount of walking, running, or standing, incorrect footwear, excess weight, or poor muscle or ligamentous tone from any cause Sometimes no reason is evident. Strain is not necessarily related to a relaxed or flat foot, it may occur when the foot appears normal and conversely be absent when it is mechanically faulty. The outstanding symptom is pain, usually confined to the inner border of the longitudinal arch, inner aspect of the ankle joint, and the metatarsal arch; occasionally it is referred to the anterior aspect of the leg and medial

aspect of the knee. It is intensified by walking and often persists for some time afterward, lameness and stiffness develop following rest The longitudinal arch may be high, normal, or low One finds tenderness of the inferior portion of the os calcis, extending along the distribution of the plantar fascia, and below the head of the first metatarsal Active and passive motions are usually normal The patient walks awkwardly and without spring, lifts the ball and heel of his foot simultaneously, and as a rule keeps his toes turned outward

Fracture of Foot. The most common are fractures of the os calcis due to direct fall on the heel, of the base of the fifth metatarsal due to twisting injury or fall, and of the metatarsal or phalangeal bones due to a direct blow

In military personnel, fracture of the neck of the second, third, or fourth metatarsal is common, presumably from abnormal strain of the anterior arch secondary to prolonged hiking and perhaps the added load of battle equipment (*march fracture*)

Edema

Bilateral Edema. The most common causes are obesity, congestive heart failure, nephritis or nephrosis, hepatic insufficiency, deficiency states, bilateral thrombophlebitis or postphlebitic syndrome, pelvic or abdominal diseases causing obstruction to blood flow through the inferior vena cava or its tributaries from the lower extremities, and processes interfering with lymph flow such as filariasis or malignant disease involving the saphenous nodes Although ordinarily most pronounced in the foot and lower legs, edema may, in certain of these disturbances, be predominant in the posterior aspects of the thighs if the patient has been long recumbent

Mild or moderate edema of the feet and ankles is frequently seen in late pregnancy, and without known cause in other normal persons—most commonly older women—especially in hot weather. It also develops in normal persons after a long period of sitting as, for example, on an overnight train or airplane ride; here, it can be prevented by keeping the feet elevated or walking about at intervals Tight garters are often a contributing factor

Puffy swelling, distinguished from true edema by failure to pit on pressure, is seen in myxedema

Unilateral Edema. This is most likely due to a local inflammatory process, unilateral impairment of lymph or venous blood flow or, rarely, hemiplegia or angioneurotic edema Lymphangitis secondary to local infection entering through a dermatophytotic fissure is perhaps the most frequent inflammatory lesion

The Skin

Eruptions. Most exanthemata and dermatoses which show an eruption on the upper extremities develop a similar, but often less striking, rash on the lower Purpuric lesions, however, are more common on the legs. The ankles and soles demand careful inspection when meningococcic infection is a possibility Sometimes in pellagra the feet and ankles are the only sites of the skin

changes although, as a rule, they show less involvement than the upper extremities The same applies to arsenical *keratosis.*

Dermatophytosis. A common and sometimes serious affliction, this occurs chiefly on the soles, toes, and interdigital surfaces Formation of minute intradermal vesicles occurs first, followed by scaling, fissuring, and, between the toes, maceration. Itching and burning are common complaints but may be absent. The disease becomes aggravated by hot weather or other cause of increased moisture Secondary infection with ascending lymphangitis is a frequent serious complication Dermatophytosis of the genitals usually spreads to the adjacent aspects of the thighs

Dermatitis. Itching, burning, fine vesiculation, redness, and local swelling may reflect sensitivity to some component of clothing such as wool, nylon or garter-metal *Winter* dermatitis is characterized by itching and variously sized and distributed areas of dryness, scaling, redness, and papulovesicular lesions *Stasis* dermatitis is common as a result of impaired return blood flow in cases of long-standing varicose veins or postphlebitic syndrome The skin of the ankle and lower third of the leg becomes infiltrated, thickened, scaly, and shows reddish or brownish discoloration Intense itching is likely, an abrasion caused by scratching often leads to ulceration Often dermatophytotic infection is a contributing factor.



Fig 34 3 Stasis dermatitis and edema of legs and feet due to post thrombophlebitic syndrome (Courtesy Dr Earl A Glicklich)

Erythema Nodosum. This is an acute or subacute inflammatory syndrome characterized by the appearance of painful tender nodules, usually on the anterior and medial aspects of the legs, much less frequently on the thighs, buttocks or upper extremities, and, rarely, on the trunk or face. Its incidence is much higher in women. Malaise, fever, sore throat and joint pains are present; often they appear several days before the nodules. The latter, symmetrically distributed and often appearing in crops, begin as rounded or oval, subcutaneous swellings 1–4 cm in diameter which usually elevate the skin and are fairly well circumscribed. At first red, they soon take on a bluish hue and pass through color changes similar to those of an ecchymosis. They disappear within a few days and never ulcerate. New lesions may continue to appear for weeks or months. In about one-quarter of patients, x-ray will show enlargement of hilar lymphnodes. Recurrences are not uncommon. The syndrome has been variously regarded as related to rheumatic infection, rheumatoid arthritis, focal infection or tuberculosis but positive proof is lacking. In this country it seems most likely to follow upper respiratory infection. Similar lesions are also encountered in some cases of coccidioidomycosis, chronic meningococcic infection and sarcoidosis, and following therapeutic use of certain sulfonamides and other drugs. Be-
, many observers regard



Fig 34 4 Localized myxedema Non pitting swelling of legs and feet with "orange peel" pitting of skin in a case of thyrotoxicosis with malignant exophthalmos White patch on right leg covers a biopsy incision (Courtesy Dr Farahe Maloof)

Localized Myxedema. This term refers to a dermatologic variant occasionally encountered in thyrotoxicosis, especially when malignant exophthalmos is present. One finds, most likely in the pretibial regions but sometimes involving large areas of the lower legs, indurated, non-tender subcutaneous plaques or nodules of varying size and shape over which the skin is pink, brown, or yellowish and has a pitted, orange-peel appearance. Inflammation or ulceration is not a feature. On pathologic examination, mucin is demonstrable in the subcutaneous tissue. In an occasional case, the abdomen or hands are involved and very rarely one finds associated clubbing of fingers.

Tuberculosis. Erythema induratum, which occurs chiefly on the calves, and is bilateral and symmetrical, has already been described (*see Chap. 3*). Ulceration may also be created by extension from tuberculosis in an underlying structure, usually bone. The lesion is painful, sluggish, irregularly shaped, has a *ragged, undermined edge, and a pallid, granulating base which secretes a watery discharge.*

Syphilis. Any of the secondary eruptions previously described as occurring on the upper extremities may be seen on the lower. Late syphilitic lesions occur chiefly on the upper half of the leg, less often on the thigh. Asymmetrically distributed, they begin as circumscribed, painless, subcutaneous nodules which gradually increase in size, become attached to the skin and later ulcerate. They tend to ulcerate in one area while healing in another, and show distinctive scar formation and circinate arrangement. Adjacent lesions tend to coalesce. The ulcer is round or oval, has clear-cut edges, and a punched-out appearance. The surrounding skin shows purplish pigmentation. Healed areas are pigmented at first but later become centrally blanched.

Post-Thrombophlebitic Ulcer. Appearing in an area of stasis dermatitis on a brawny edematous leg, this is usually initiated by mild trauma. Careful questioning should elicit a story consistent with an episode of deep thrombophlebitis at some time in the past. The ulcer almost always develops on the lower third of the leg, usually just above the internal, less often the external malleolus. Varying in diameter from a few millimeters to 10 or more centimeters, it is painful, long-standing, sluggish, superficial, irregular, and has an unhealthy edge and granular base. A zone of infiltrated, scaly, thickened, reddish, or brownish skin surrounds it. After healing, a brownish scar remains.

Varicose Ulcer. In general this has the same characteristics as post-thrombophlebitic ulcer. A varicose vein is traceable downward into the ulcerated area. Most so-called varicose ulcers are actually post-thrombophlebitic. A varicosity is indictable only when post-thrombophlebitic syndrome can be excluded by history and absence of the typical brawny edema. In the presence of the latter, varicosities are usually secondary to impaired circulation in the deep veins and hence, *per se*, not responsible for ulceration.

An ulcer on the leg above its mid-portion is rarely, if ever, due to venous stasis.

Perforating Ulcer. The result of a neurotrophic disturbance, this is most commonly seen in diabetes mellitus and tabes dorsalis. Starting as a callus at a



FIG 34 5 Bilateral edema, stasis dermatitis, and extensive ulceration in post-thrombophlebitic syndrome (Courtesy Dr Robert R Linton)

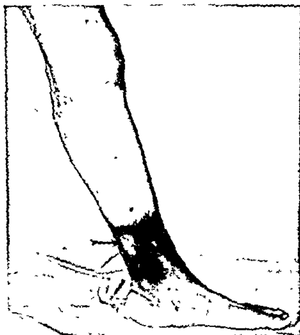


FIG 34 6 Stasis dermatitis with ulceration (arrow) due to long-standing varicosities In contrast to post thrombophlebitic syndrome, edema is minimal to absent (Courtesy Dr Earl A Glicklich)



FIG 34 7 Perforating ulcer of foot in diabetes Arrow points to crater Light-colored zone surrounding it is callus

pressure point on the foot, usually the first or fifth metatarsophalangeal joint, it soon breaks down beneath the horny layer The callus is cast off, leaving a suppurating ulcer which extends deeply into the underlying tissue, sometimes even involving bone The ulcer is called *perforating* because of its depth and stubborn progress in spite of therapy which should be effective, actual perforation of the foot is rare In diabetics this lesion is often incorrectly attributed to arterial disease when actually the circulation is not seriously impaired

Callus. A hard, thickened, usually painful overgrowth of corneum develops at a pressure point on the foot or a toe When it is brown or yellowish, dermatophytosis is suggested.

Plantar Wart. Occurring singly or multiply on the sole, this appears at first glance to be a simple callus, but inspection following paring away of the superficial horny layer will reveal brownish pinpoint dots, filiform segments, and sometimes moisture Such lesions are very painful.

Epidermoid Carcinoma This is relatively rare on the lower extremity except about a scar or the opening of a persistent sinus

Malignant Melanoma. The tumor starts as a small, deeply pigmented mole in the skin, sometimes beneath a toenail.

THE JOINTS

The various types of arthritis characterized by local swelling, redness, tenderness, limitation of motion, deformities, and atrophy of regional muscles result from the same causes which produce them in the upper extremities.

LOCALIZED SWELLING

This may be due to benign or malignant tumor, syphilis, tuberculosis, or other inflammatory process of bone or soft part. Bone swelling may be a local manifestation of a systemic disease such as osteitis fibrosa generalisata or Paget's disease. Unless the diagnosis is obvious, pain or swelling in an extremity demands x-ray study; this may establish diagnosis but exploration is often necessary.

Prepatellar Bursitis. Popularly known as *housemaid's knee*, this is marked by swelling with or without tenderness anterior to the patella, the knee joint is not involved. Infection may develop, giving fever and leukocytosis

Subcutaneous Nodules. The characteristic lesions of rheumatic infection and rheumatoid arthritis may be found in the tendons of the patella, external malleolus or dorsum of the foot The distribution of gouty tophi is similar, the large toe is a common site Careful palpation along the peripheral vessels of the lower extremities may reveal the small, hard, shotty lesions sometimes found in polyarteritis nodosa

Ganglion. Favored sites are the anterior and lateral aspects of the ankle, dorsum of foot, and popliteal space

Osteomyelitis. The disease is most likely to arise in the upper, middle or lower third of the femur, or lower third of the tibia. Onset is sudden and attended by pain, swelling, extreme local tenderness, fever, and leukocytosis Early x-ray examination is usually negative, later, it may show elevation of the periosteum and eventually bone destruction *Brodie's abscess*, a mild, localized osteomyelitis caused by staphylococcus of low virulence, typically occurs in the lower third of the tibia Onset is insidious and accompanied by moderate pain and tenderness Systemic reaction is slight and the course often chronic The characteristic x-ray picture is of a clean-cut cavity in bone surrounded by a zone of increased density Low-grade osteomyelitis of a phalanx secondary to infection extending from a superficial lesion such as a paronychia is sometimes encountered.

Tuberculosis. With the exception of the spine, the hip is the most common site of joint tuberculosis. Limp, pain often referred to the knee, and later, restriction of motion, abnormal position, and shortening develop. Swelling may be absent, or be evident in the groin or inferior gluteal region Sinus formation occurs in the advanced stage X-ray reveals a destructive process in the neck and head of the femur, and in the acetabulum

The knee is the next most frequent site. Limp, swelling, increase of local heat, pain, and tenderness occur first Muscle atrophy, flexion deformity, and shortening may develop Late complications are subluxation of the tibia on the femur, and sinus formation X-ray shows extensive bone destruction with little new bone formation Involvement of an ankle is marked by comparable manifestations

Syphilis. Periostitis and osteitis of the tibia are not uncommon in the congenital, and late acquired forms Pain, often worse at night, local tenderness, and usually some swelling are present. The tibia feels thickened, nodular and

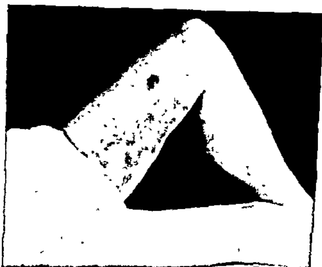


Fig 34 8 Subcutaneous hemorrhage of thigh, probably arising in the muscles, in a case of scurvy

may show anterior bowing (*saber-shin*) Gumma of bone is rare but occasionally occurs as a centrally placed destructive process Found in association with active or quiescent tabes dorsalis, *Charcot joint* is most common in the knee (*see* Chap 35). It is characterized by painless, pronounced swelling, relaxation of ligaments, weakness, usually evidence of increased fluid in the joint, and, often, subluxation.

Scurvy. Hard, painful, tender swelling, especially in the calf, may be encountered in scurvy as a result of intramuscular hemorrhage. Sometimes it is the first indication of trouble Exquisitely tender swelling over the tibia or femur can occur from subperiosteal bleeding

Benign Tumor of Soft Parts. Lipoma. Lipoma appears as a soft, lobulated, subcutaneous growth. Occasionally the tumor is deeply situated between the muscles and can then be recognized only by x-ray.

Neurofibroma (von Recklinghausen's Disease). This is marked by small, multiple, freely movable, perhaps slightly tender subcutaneous nodules situated along the nerve trunks. Freckle-like and larger spots of pigmentation are almost always found distributed over the body. A diffuse, often symmetrical form appears as soft, perhaps pigmented folds of skin and subcutaneous tissue. Neurofibroma arising in the sciatic or other large nerve trunk can attain considerable size and may become malignant (*neurogenic fibrosarcoma*)

Malignant Tumor of Soft Parts. Sarcoma. Sarcoma of the neurogenic or fascial type, the most common malignant tumor of the soft parts, is a fusiform, firm growth deeply situated in the muscles of the thigh. Liposarcoma, malignant synovioma, and rhabdomyosarcoma may also occur. Biopsy is necessary for diagnosis.

Benign Tumor of Bone. Osteoma, Chondroma, and Osteochondroma. These are non-malignant tumors most often seen about the knee joint. A mixed growth is not uncommon. Cartilagenous exostoses tend to be multiple. The tumors

appear as hard, irregular, symptomless growths connected with underlying bone One form which is encountered beneath the nail of the great toe causes excruciating pain It is often incorrectly diagnosed as a malignant tumor. X-ray confirms diagnosis.

OSTEOID OSTEOMA. This is a painful localized tumor usually involving the shaft of a long bone, which presents a characteristic x-ray appearance that distinguishes it from a malignant process.

EOSINOPHILIC GRANULOMA. This may occur in a long bone of the leg; it can be diagnosed with certainty only by pathologic examination.

BENIGN GIANT CELL TUMOR. Benign giant cell tumor arises in the epiphyseal ends of the long bones, most commonly about the knee. It causes pain and a palpable mass X-ray shows a central, expansile bone-destroying growth without new bone formation.

Malignant Tumor of Bone. OSTEOGENIC SARCOMA. Osteogenic sarcoma is most likely to start at the lower end of the femur or upper end of the tibia Constant pain, unrelieved by resting the part, is the first symptom It is often referred to the joint Early physical examination shows little; later, one finds a fusiform



FIG 349 Osteogenic sarcoma, distal end of femur Increased bone density surrounding an irregular area of destruction



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Gangrene. Barring frost-bite or other serious traumatic insult, gangrene beginning in one or more toes and often spreading to the foot, is most likely due *to impaired arterial blood flow;* here it may be initiated by mild trauma. The part will be initially white, later reddish to purple and eventually black, becomes shriveled and, when well established, shows a sharp line of demarcation between affected and unaffected tissue (*dry gangrene*). When infection is a factor, as is often the case, especially in the diabetic, redness and swelling appear first, followed by color changes just noted, serous oozing, and eventually ulceration and necrosis (*moist gangrene*).

[illegible]

to relaxation of the anterior arch, faulty footwear and localized degenerative changes of the plantar nerve. Removal of the shoe gives temporary relief; cure is often effected by use of proper footwear and treatment of the mechanical variant

Erythromelalgia. This term refers to attacks of pain in the extremity, most often the ball of the foot and large toe It predominates in men. The pain is intense and described as an acute sensation of burning or sticking. Redness, tenderness, and local heat are evident In contrast to Raynaud's disease, attacks are induced or aggravated by heat The cause is not clear; it is thought that erythromelalgia is not a separate entity but related to some vascular or neurotrophic disturbance Acute gout must be excluded.

Meralgia Paresthetica. An area of hypesthesia, paresthesia or hyperesthesia, with or without pain, develops on the anterolateral aspect of the upper thigh, *uni- or bilaterally. It is presumably due to irritation of the external cutaneous* nerve and significant chiefly because it may be confused with the signs of a more serious neurologic disorder

Tender Toes. These occur during the late stages of typhoid fever, probably as a result of neuropathy In subacute bacterial endocarditis, an embolus blocking a small vessel in a toe produces a deep red or purplish spot which is tender; it lasts only a few days.

Enlargement of the extremities, clubbing of the digits, dactylitis, splinter hemorrhages, and other variants have the same significance as in the upper extremities (*see* Chap 6)

THE ARTERIES

Raynaud's Disease. Vasomotor phenomena similar to those occurring in the fingers (*see* Chap 6) are encountered in the toes but, as a rule, are much less striking Episodes are most likely to occur in persons whose feet are persistently cold and clammy; the process is usually bilateral and symmetrical, the same digits being involved in each attack The patient will complain of coldness of one or more toes on each foot; on examination, these will appear pallid or slightly cyanotic, more moist, and colder to the touch than the others. Pronounced color

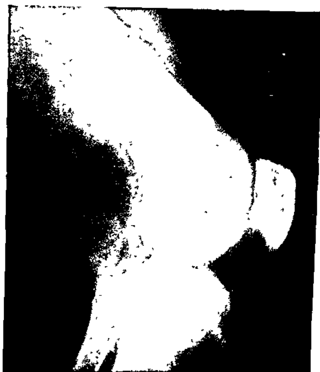


FIG 34 10 Metastatic lesion of femur from carcinoma of bronchus Bone destruction is predominant, proliferation, minimal

mass and some limitation of joint motion The diagnosis should be suspected in a child or young adult presenting this picture, it can be confirmed by x-ray.

CHONDROSARCOMA This is marked by growth of a large lobulated tumor often in the vicinity of the pelvis, it occurs in the older age group and has a better prognosis than osteogenic sarcoma.

EWING'S SARCOMA Often mistaken for osteomyelitis, Ewing's sarcoma arises in the shaft of a long bone, especially the femur or fibula Pain and palpable tumor, accompanied by localized heat, and redness, and perhaps slight elevation of temperature and leukocyte count are characteristic Diagnosis is established by x-ray

METASTATIC DISEASE. Metastatic disease must always be considered in any bone tumor Sometimes a lesion is discovered well in advance of the primary growth In the adult, carcinoma of renal parenchyma, prostate, lung, thyroid, or breast is especially disposed to metastasize to bone, in youth, neuroblastoma Malignant lymphoma and reticulum cell sarcoma are occasionally encountered in the bones of the lower extremities

MISCELLANEOUS LESIONS

Tenosynovitis of Achilles Tendon. This causes pain in the posterior aspect of the ankle and heel, tenderness, and often, palpable crepitus over the tendon when the foot is moved up and down.

should be confirmed by the sphygmomanometer placed at varying levels of the extremity. If pressure in the machine is maintained at a point between the systolic and diastolic levels, the degree to which the mercury column oscillates will indicate the volume of pulsation in the underlying arteries No oscillation at any point means that blood flow through the major vessels at that level is totally or almost completely obliterated Some physicians prefer to use the oscillometer, a more elaborate and accurate device which operates on the same principle, but for ordinary purposes, sphygmomanometric observations are sufficiently dependable

The average case of arteriosclerosis will show obliteration of the dorsalis pedis and posterior tibial pulses; the artery walls may or may not be palpable *In the severe case,* all of the major arteries are likely to be diffusely or segmentally obstructed, with consequent absence of pulsations. Thickening, tortuosity and calcification of the walls of the smaller arteries are not as apparent in the lower extremities as in the upper, but are readily palpable in the larger vessels Calcification may be discovered by x-ray but this finding is of relatively little importance since the degree of circulatory impairment does not parallel that of the calcification

When blood-flow is seriously hampered, the foot feels cold to the touch and shows thinning, atrophy or even shrivelling, and loss of hair and subcutaneous tissue The toenails are often ridged and thick Color changes incident to change of position are diagnostically important· in the normal person, the foot can be raised or lowered to any level with respect to the heart without undergoing appreciable color variation; in obliterative disease, it will blanch when elevated and develop rubor or cyanosis when dependent, the speed and degree of these alterations being indices of severity Provided varicose veins are not present, the rate of filling of its superficial dorsal veins, when the foot is lowered following a period of elevation, can also be used as an index of arterial efficiency normally these vessels, which are barely visible when the foot is elevated, will fill and become clearly visible within 10 seconds following lowering; if filling time is greater than 20 seconds, pronounced arterial impairment can be assumed

Gangrene, occurring spontaneously or following mild trauma or ill-advised minor surgical procedure, is a common and serious complication (*see above*)

Especially in older persons with or without diabetes, and sometimes in a younger diabetic, one encounters a form of arteriosclerosis which is marked *more by thickening and calcification of the media than of the intima (Monckeberg arteriosclerosis)* Impaired blood flow and eventual gangrene are likely.

Since it has been well established that a certain number of patients with claudication or other indication of impaired blood flow have segmental arterial obstruction, arteriography has become increasingly important in the study of occlusive disease If x-ray following injection of an opaque medium shows localized block, it may be possible, by means of an arterial graft, to re-establish blood flow sufficiently to alleviate symptoms or obviate need for amputation.

Thrombo-angiitis Obliterans As indicated earlier (*see* Chap 6), Buerger's disease is a generalized proliferative disturbance of the arteries and veins which

changes seen in the fingers are rarely observed. Such manifestations of impaired blood flow as absence of arterial pulsation, color variations induced by elevation and dependency, and trophic changes, ulceration, and gangrene will not develop unless there is a coexistent obliterative arterial disease.

Arteriosclerosis. This is common in both sexes beyond 50; in diabetics it often occurs much earlier. The clinical picture depends on the relationship between *the degree of damage to the main vessels and the adequacy of collateral circulation* For example, even though blood flow through a major artery is seriously impaired, either throughout its course or segmentally, symptoms will be absent *if collateral circulation is well established*, but severe trouble will ensue if the latter is inadequate Although the arteriosclerotic changes are bilateral, symptoms and signs usually appear earlier and continue more advanced in one extremity than the other. The usual early symptoms of insufficient blood flow are *coldness of the foot with perhaps attacks of burning usually* worse at night, and intermittent tingling or other form of paresthesia, especially in the anterior part of the foot These are aggravated by heat and diminished by keeping the extremity dependent. If circulation is impaired at the level of the knee or higher, the patient will have experienced, *prior to onset of these symptoms*, intermittent claudication—cramp-like pain in the calf, or, less often, the thigh, precipitated by walking and immediately relieved by standing still. He may also have had *spasmodic pain when recumbent, especially in the early morning hours, with* relief by lowering the legs, this symptom can often be prevented by sleeping with the head of the bed elevated so that the body is inclined downward In the individual case, the intensity of pain, and distance and speed of walking required to provoke it remain fairly constant and are a good index of the severity of the process If ulcer or gangrene develops, the pain it creates overshadows the claudication.

On examination, the earliest sign is diminution or absence of pulsation in the peripheral vessels but symptoms may exist even in the presence of palpable pulses The *dorsalis pedis* pulse should be felt for with the patient in a comfortable position and his leg and foot completely relaxed One's fingers should be rested lightly on the dorsum of the foot and the artery first felt for just lateral to the tendon of the extensor hallucis, at the level of the proximal end of the first metatarsal bone If no pulsation is felt here, the adjacent areas must be *similarly palpated since the artery often* pursues an aberrant course Only when careful palpation of the upper half of the dorsum has been unrewarding, can absence of dorsalis pedis pulsation be assumed The *posterior tibial* pulse is felt deep in the popliteal space. Since it can be readily overlooked unless surrounding tendons *and muscles are well relaxed, it is best to* have the patient lie prone with his thigh on the table and his leg held partially flexed by a third person The *femoral* pulse is felt for just below the mid-portion of Poupart's ligament, with the patient *supine and the extremity* extended.

Presence or absence of pulsation, if not clearly evident by palpation, can and

of incapacity; earlier aches or pains may have been attributed to foot strain or other orthopedic disorder. In arteriosclerosis, incapacity is likely to follow initial symptoms by only a few months.

5. Small areas of transitory thrombophlebitis appearing here and there in the superficial veins of one or more extremities are not uncommon; when present, these are virtually pathognomonic of Buerger's disease

6 Secondary infection, when present, is likely to remain confined to the foot whereas in arteriosclerosis there is a tendency to a more rapid and diffuse spread.

Peripheral Embolism. As in an upper extremity, peripheral embolism is usually found at a major bifurcation Excruciating pain in the extremity, pallor with or without blotchy cyanosis, absence of arterial pulsation, palpable coldness, impaired sensation, and some degree of paralysis are the chief manifestations Especially in the bedridden patient, pain may not be striking, but he will complain of numbness or other paresthesia The changes indicated are found distal to the point of obstruction but, except for absence of pulsation, do not appear as high as the anatomic site of the occlusion since collateral circulation will supply blood for a considerable distance below it Sometimes transient minor attacks of numbness or coldness will appear days or weeks prior to the occlusive episode These are regarded as due to small fragments breaking away from the source, in advance of the major embolus

A similar picture can be created by thrombotic occlusion of an artery. The differentiation between this and peripheral embolism is often difficult. Well-established arteriosclerosis or thrombo-angiitis obliterans favors the former; a situation such as mitral stenosis, auricular fibrillation or recent myocardial infarction predisposing to peripheral embolism, the latter However, too much reliance cannot be placed on the presence of a cause for embolism since poor general circulation, based on myocardial insufficiency which might well accompany one of the cardiac disturbances just mentioned, will predispose to thrombus formation in a diseased artery Deep thrombophlebitis and peripheral embolism can also be confused The more important differential points are that in embolism, the peripheral pulses are obliterated and the part blanched and cold, whereas in phlebitis, the pulses may be present and the part is more likely warm and perhaps slightly cyanotic These distinctions are not entirely dependable since, rarely, acute thrombophlebitis will cause vasospasm of such magnitude as temporarily to obliterate the peripheral pulses and create the picture of local ischemia.

Unless a major occlusive insult is recognized promptly and can be surgically treated, gangrene distal to the point of effective collateral circulation will be the end result.

Trench Foot. Common in soldiers restricted to trenches or foxholes for prolonged periods, trench foot results from local vascular changes secondary to prolonged exposure of the part to cold and moisture, especially when dependency, immobility, and perhaps constriction by a shoe or clothing are also operative The same phenomenon occurs in persons long confined to lifeboats



FIG 34 11 Gangrene of toe due to arteriosclerosis (Courtesy Dr Robert R Linton)

begins in early or mid-adulthood, is rarely encountered in women, usually predominates in the lower extremities, and often exists for years before causing serious trouble. Although relatively common in the Jewish race, it is, contrary to some teaching, by no means confined to this group When the lower extremities are involved, the manifestations, dependent on impaired blood flow, are comparable to those encountered in arteriosclerosis. One can usually distinguish between the two diseases by keeping in mind the following features of thrombo-angiitis obliterans.

1 Women are rarely aff-
2 The disease begins i — than in later years
3 The vessel walls lack — and calcification demonstrable by x-ray is absent or —
4. As a rule, progress is slower and complications develop late. A carefully taken history will show that some symptoms existed for years prior to the stage



FIG 34.12 Arteriogram of left thigh showing arteriosclerotic occlusion of superficial femoral artery. Arrow points to beginning of obstructed segment Collateral filling of the artery distal to involved segment is apparent.

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1. Women are rarely affected.

2 The disease begins in youth or middle-age rather than in later years

3 The vessel walls lack the rigidity or beading characteristic of arteriosclerosis, and calcification demonstrable by x-ray is absent or minimal

4. As a rule, progress is slower and complications develop late A carefully taken history will show that some symptoms existed for years prior to the stage



Fig. 34 12 Arteriogram of left thigh showing arteriosclerotic occlusion of superficial femoral artery Arrow points to beginning of obstructed segment. Collateral filling of the artery distal to involved segment is apparent.

Depending on stagnation, and appearing only after valvular insufficiency has reached the point of permitting reverse flow of blood when the patient is upright, the important manifestations in order of their appearance are.

1 Prominence, increased caliber, and tortuosity of the impaired veins. Although these changes below the knee are invariably apparent to both the examiner and patient, they may be obscured in the thigh by a thick layer of subcutaneous tissue. In this event, distention of the internal saphenous trunk in the thigh can be detected by the following procedure: With the patient standing, one briskly flicks or taps the prominent vein below the knee while, with the fingers of his other hand, lightly palpating over the saphenous opening in the groin; if a varicosity is present an impulse will be transmitted upward to the palpating fingers by the tense column of blood, whereas over normal vein no impulse will be felt.

2. Sensation of heaviness in the legs and easy tiring when standing. The patient rarely complains of pain; if he does, one should look for concomitant arterial disease, foot strain, or other cause.

3 Pigmentation about the ankle in the region of the involved vein

4. Stasis dermatitis characterized by itching, redness, thickening, scaling, brownish discoloration, and often superficial oozing of serum Dermatophytosis is frequently a contributing factor to these changes

5 Edema Although there may be slight swelling about the ankles, edema is not striking If one finds it extending upward toward the knee or higher, impairment of deep veins due to previous thrombophlebitis is likely

6. Varicose ulcer Ulceration may result from varicosity alone but as a rule it is more apt to be a reflection of deep venous impairment (*see above*)

The *Trendelenburg test* is performed to determine the degree of circulatory impairment and confirm the reversal of flow in the affected vessels With the patient supine, the leg is held elevated to an angle of 45°, after a few moments, the superficial veins will collapse A tourniquet is then applied to the upper thigh just tightly enough to constrict the veins and the patient is instructed to stand If the veins do not fill with the tourniquet in place but fill immediately *from above downward* when the tourniquet is removed, reversal of flow and incompetency of valves is indicated If, with the tourniquet still in place, the veins fill partially *from below upward* there is incompetency of valves and reversal of flow in the communicating veins between the deep and superficial systems, if the tourniquet is then removed, and the partially filled veins become more distended, both the main trunks and the communicating branches are incompetent By repeating the test with the tourniquet at various levels of the extremity or applying multiple tourniquets simultaneously at different levels and releasing one after another the location of incompetent perforating veins can be accurately determined This refinement is not necessary for ordinary clinical purposes

Because of stasis, varicose veins are particularly subject to thrombophlebitis and are sometimes the source of *small* pulmonary emboli. (Large emboli starting in the legs are usually from the deep veins.)



FIG 34 13 Extensive gangrene, legs and feet, due to saddle embolus at aortic bifurcation in a case of rheumatic heart disease with auricular fibrillation Insult occurred 1 week earlier (Courtesy Dr Robert R Linton)

(*immersion foot*) The severity of the process is dependent on duration of confinement, and degree of such factors as cold, immobility, general exposure with consequent loss of body heat, dehydration, and nutritional deficiency.

During the period of exposure the patient experiences coldness, numbness, tingling, and aching or crampy pain in the feet, ankles, and perhaps lower legs As a rule, the skin locally is pallid with mottled cyanosis, or definitely purplish, and often anesthetic; edema is not pronounced Following return to favorable environment, the course is extremely variable The mildest case will recover completely following a few days of numbness, coldness, and stiffness Where damage was more serious one will find pain (sometimes severe), anesthesia, hyperesthesia or paresthesia, redness, local warmth, edema, bleb formation, ecchymosis or perhaps massive blood extravasation, and impairment of motor control Gangrene sometimes develops Recovery is usually slow In all but the mild cases, pain, sensory disturbances, and perhaps impaired locomotion will persist for months or years, Raynaud's phenomena sometimes develop.

THE VEINS

Varicose Veins. Dilatation, frequently accompanied by tortuosity of the superficial veins in one or both lower extremities, is common in adults, particularly women. It tends to run in families The underlying cause is congenital weakness of the valves of the affected veins, pregnancy, obesity, and occupations requiring long periods of standing are often contributing factors but only if potential valvular weakness is present. The internal saphenous system is the most frequent site, although external saphenous involvement is not uncommon.

or prostate may be complicated by thrombophlebitis of the regional veins but often clotting here is blamed for a pulmonary embolus when actually the latter had its source in a leg vein.

Thrombophlebitis of a *superficial* vein is marked by relatively slow onset—12 hours or more—and mild symptoms, pain is minimal Tenderness over the vein usually appears first, accompanied by a slight rise in temperature (99°–100°) lasting about 3 days The skin over the involved segment is red, swollen and tender, edema of the leg itself is rare The acute phase usually subsides within 10 days but tenderness may persist for some weeks, and as a result of thrombus organization and fibrosis of the vessel, the latter becomes firm and cord-like The process is sometimes initiated by irritation at the site of an intravenous injection Migratory thrombophlebitis occurring in thrombo-angiitis obliterans has already been discussed (*see above*)

Thrombophlebitis of a *deep* vein is characterized by sudden onset of pain, usually in the calf, sometimes behind the knee or along the inner side of the thigh or groin. For the first week or so, fever is higher (100°–102°) than in superficial phlebitis and often continues at a lower level for some weeks longer Tenderness along the course of the deep veins, especially in the calf, and brawny edema increased by dependency also develop Edema reaching into the thigh indicates extension into the iliac vein. The extremity is pallid or cyanotic, and pulsation in the peripheral arteries may be diminished, presumably as a result of secondary spasm. Pain in the calf may be increased, or if not present, be elicited by forced dorsiflexion of the foot (*Homan's sign*) The tenderness may last for weeks. The swelling usually subsides within 2–6 weeks, although some

up with post-thrombophlebitic syndrome (*see below*)

At times the process is by no means as acute as just described, the manifestations, being less striking, may be detectable only by careful examination *Under any of the circumstances noted above as predisposing to its development, this complication must always be watched for, especially in the presence of low-grade fever not otherwise explainable, if it is discovered, proper treatment may avert serious pulmonary embolism*

Post-Thrombophlebitic Syndrome. Following an attack of deep thrombophlebitis, especially if untreated, the end-result may be chronic permanent impairment of venous circulation As a rule, the diseased vein recanalizes, but since its valves were destroyed, return blood-flow is inadequate when the patient is upright. The resultant increased venous pressure is imparted to the communicating veins which then lose their valvular efficiency because of dilatation due to the added load and their lack of supporting structures. Blood flow through the communicating veins is now from the deep to the superficial system—the reverse of normal This change, in turn, by adding a greater load and increasing pressure in the superficial system creates dilatation, valvular inadequacy, and even-



and veins unfilled Slight ... d and marked filling from above indicates incompetence of valves of long saphenous vein

Thrombophlebitis. The blood in a vein becomes clotted and there is an accompanying low-grade inflammation of the vessel and its associated lymphatics The leg is by far the most common site, often the process starting here will extend upward into the thigh, and sometimes into the iliac vein or even the vena cava The etiology is not clear but it is generally accepted that stagnation, as from prolonged standing, long confinement to bed, or impaired general circulation, changes in the blood which favor clotting, such as increased thrombokinase or reduced antithrombin, and perhaps changes in the endothelial lining of the vessel are operative Infection, obesity, debility and dehydration are often, but not necessarily, contributing factors The most common circumstances favoring development of thrombophlebitis in a leg are listed below; the older the patient, the more likely the complication.

1 Local injury, infection or operation
2 Operation elsewhere, especially within the abdomen.
3 Post-partum state
4. Congestive heart failure.
5 Debilitating disease
6 Carcinoma, especially of the pancreas
7 Severe general infection, such as pneumonia or typhoid fever.

Infection of, or operation on a structure within the pelvis such as the uterus

or prostate may be complicated by thrombophlebitis of the regional veins but often clotting here is blamed for a *pulmonary embolus when actually the latter* had its source in a leg vein.

Thrombophlebitis of a *superficial* vein is marked by relatively slow onset—12 hours or more—and mild symptoms, pain is minimal Tenderness over the vein usually appears first, accompanied by a slight rise in temperature (99°–100°) lasting about 3 days. The skin over the involved segment is red, swollen and tender, edema of the leg itself is rare. The acute phase usually subsides within 10 days but tenderness may persist for some weeks, *and as a result of thrombus organization* and fibrosis of the vessel, the latter becomes firm and cord-like The process is sometimes initiated by irritation at the site of an intravenous injection Migratory thrombophlebitis occurring in thrombo-angiitis obliterans has already been discussed (*see above*).

Thrombophlebitis of a *deep* vein is characterized by sudden onset of pain, usually in the calf, sometimes behind the knee or along the inner side of the thigh or groin. For the first week or so, fever is higher (100°–102°) than in superficial phlebitis and often continues at a lower level for some weeks longer Tenderness along the course of the deep veins, especially in the calf, and brawny edema increased by dependency also develop Edema reaching into the thigh indicates extension into the iliac vein The extremity is pallid or cyanotic, and pulsation in the peripheral arteries may be diminished, presumably as a result of secondary spasm. Pain in the calf may be increased, or if not present, be elicited by forced dorsiflexion of the foot (*Homan's sign*) The *tenderness may* last for weeks The swelling usually subsides within 2–6 weeks, although some residual, brawny enlargement of the leg may persist for several months During the acute stage, the white count is moderately elevated and blood sedimentation rate rapid The latter is a good index of activity since it remains elevated until the process has healed If complete recovery does not occur, the patient will end up with post-thrombophlebitic syndrome (*see below*)

At times the process is by no means as acute as just described, the manifestations, being less striking, may be detectable only by careful examination. *Under any of the circumstances noted above as predisposing to its development, this complication must always be watched for, especially in the presence of low-grade fever not otherwise explainable, if it is discovered, proper treatment may avert serious pulmonary embolism*

Post-Thrombophlebitic Syndrome. Following an attack of deep thrombophlebitis, especially if untreated, the end-result may be chronic permanent impairment of venous circulation As a rule, the diseased vein recanalizes, but since its valves were destroyed, return blood-flow is inadequate when the patient is upright The resultant increased venous pressure is imparted to the communicating veins which then lose their valvular efficiency because of dilatation due to the added load and their lack of supporting structures Blood flow through the communicating veins is now from the deep to the superficial system—the reverse of normal This change, in turn, by adding a greater load and increasing pressure in the superficial system creates dilatation, valvular inadequacy, and even-

tually a varicose status of the superficial veins. The leg becomes brawny, edematous, and usually shows brownish discoloration, especially on the inner aspect of its lower third, predominant just above the internal malleolus Ulceration here is common; because of the evident superficial varicosity, it is often wrongly diagnosed as varicose ulcer.

Phlebothrombosis. This term is applied to thrombosis of a deep vein in a lower extremity which develops insidiously and shows few or no clinical manifestations. In general it occurs under the same circumstances as thrombophlebitis but is for two reasons more dangerous. (1) Because of the paucity of local manifestations its existence is not suspected until a major embolic episode occurs (2) The clot is less adherent to the vessel wall so that the danger of embolism is correspondingly greater

The indications of deep thrombophlebitis described above are minimal or absent. In any situation in which this complication is likely, careful day-to-day examination of the patient for the signs indicated below should be carried out in the hope of detecting the process and initiating treatment before massive embolization occurs. One should be particularly alert in the presence of slight fever, pulse rate out of proportion to temperature, pain in the foot, calf, or popliteal space however slight, or any variant suggesting small pulmonary embolism such as sudden rise in temperature, pulse or respiration, short episode of faintness or syncope, cough, or pleural pain

On examination one looks for the following signs, any of which will lend support to the diagnosis of phlebothrombosis

1. Creation of pain (or aggravation if it is already present) in the calf or popliteal space by dorsiflexion of the foot while the leg is extended

2 Tenderness along the course of a deep vein In the calf or popliteal space, this is best detected by pressing deeply into the posterior structures toward the posterior aspect of the tibia, in the thigh, by pressing into its inner surface toward the femur

3 Edema Rarely pronounced, this may be evident as fulness or loss of normal ridging of the dorsum of the foot, or be detected by careful day-to-day circumferential measurements of the largest part of the calf and the smallest part of the ankle.

4 Prominence of veins on the dorsum of the foot, with the patient supine

5. A sensation, on palpation, of increased tenseness or fulness of the calf muscles

6. Appearance of a thrombosed area in a superficial vein, especially on the back of the leg In the absence of a varicosity which predisposes to thrombophlebitis, involvement of a segment of a superficial vein suggests extension through a communicating vessel from a process in the deep system.

Minimal pain, tenderness or edema in one leg can sometimes be brought out by comparison with the other, but it must be remembered that failure to detect differences does not exclude the possibility of phlebothrombosis because it is so often a bilateral phenomenon.

Whenever, under pertinent circumstances, thrombus formation in a deep vein

is regarded as at all likely, serious consideration must always be given to anticoagulant therapy or vein ligation in order to reduce the chance of serious and perhaps fatal pulmonary embolism

THE LYMPH VESSELS

Acute Lymphangitis. Characterized by red streaks along the course of the lymphatics leading from an acute infectious process, and accompanied by enlargement and tenderness of the regional lymphnodes, this is usually caused by local streptococcic infection Systemic reaction is usually severe, unless antibiotic and local therapy is promptly initiated. Acute lymphangitis is a common complication of dermatophytosis

Elephantiasis. Extreme chronic swelling and induration of one or both lower extremity and accompanied by similar swelling of the genitalia, is occasionally seen.

THE NAILS

The toe nails may show changes of the same nature and significance as those observed in the fingernails (*see* Chap. 6).

35

THE JOINTS

Pain, subjective stiffness, local muscular weakness, and restricted or exaggerated mobility are the *predominant complaints in disease of the joints* The pain may vary from mild discomfort on use to agony present even with the part at rest and making all motion impossible Stiffness may be present constantly or be noticed only after a period of rest, with disappearance after "limbering up" Restriction may vary from slight interference with function to complete fixation Sometimes the patient will seek advice because of some more objective manifestation, such as swelling, redness, tenderness, distortion, or coldness or excessive sweating of an extremity reflecting a vasomotor component.

At times the above indications will be less prominent than systemic symptoms such as fatigue, anorexia, weight loss, fever, or other variant attributable to the underlying disease as, for example, headache due to arthritis of the cervical spine, or the lacrimation and photophobia of uveitis complicating rheumatoid arthritis

EXAMINATION OF JOINTS

Examination of the joints—by inspection, palpation, and tests for range of motion—should always be performed systematically The physician should train himself to follow a specific routine, beginning with the cervical, thoracic and lumbar segments of the spine, then proceeding to the jaw, shoulder girdle, upper extremities, pelvic girdle, and lower extremities Inspection and palpation are carried out together

Inspection and Palpation

Enlargement. This is unmistakable, except in the presence of pronounced muscular atrophy or loss of flesh which may cause a normal-sized joint to appear enlarged. *Bony-hard* enlargement indicates bony or cartilaginous proliferation; *boggy* enlargement, infiltration or thickening of capsular and periarticular structures, *fluctuant* enlargement, fluid in the joint To test for fluctuation, one places his forefingers a few centimeters apart on the swollen area, and alternately presses briskly with one while lightly palpating with the other, if an impulse can be transmitted back and forth between the two fingers, fluid is almost invariably present Occasionally pronounced edema of the tissues surrounding the joint will impart a similar but less striking sensation. A *floating*

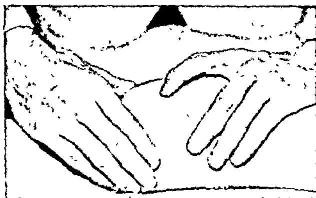


Fig 35 1. Proper position of fingers in examination for floating patella

patella is the best sign of knee-joint effusion To demonstrate this, the lateral and anterior surfaces of the joint are compressed between the examiner's hands to limit escape of fluid in any direction, and the patella is then briskly pressed posteriorly with one finger: if the patella is heard or felt to knock against the femur and rebounds as pressure is released, an abnormal amount of fluid is present.

Abnormality of Contour. This can usually be readily recognized by comparison with the corresponding joint, or with what one knows from experience to be normal Effusion may result in loss of natural depressions around the joint or, if large, cause actual bulging. A bony overgrowth may be obvious as in the case of a Heberden's node but if within the joint may be manifested only by sudden arrest of free joint motion at a certain point.

Distortion and Malposition. Muscle spasm or contracture, destruction or proliferation of joint surfaces, or relaxation of supporting ligaments will create these changes. Partial dislocation (*subluxation*) occurs with pronounced abnormal muscular pull, or relaxation of ligaments

Signs of Acute Process. Redness and increased surface temperature when combined with tenderness and swelling usually indicate an acute disturbance.

Muscular Atrophy. This may be due to disuse, associated neurotrophic disturbance, or changes in the muscle occurring as an integral part of the underlying disease The affected part is shrunken, flaccid, and shows impaired function.

Neurotrophic Disturbances. Indications are lowered skin temperature, excessive sweating, mottling, cyanosis or pallor, glossiness and atrophy of the skin around a joint, and sometimes atrophy of regional muscles

Sinus Usually this leads to an abscess in or near the joint, necrosed bone, or, rarely, to a gouty tophus It is most often due to tuberculosis or chronic osteomyelitis

Shortening. The more common causes are bone or joint destruction from an infectious process such as tuberculosis or osteomyelitis, dislocation, over-riding

35

THE JOINTS

Pain, subjective stiffness, local muscular weakness, and restricted or exaggerated mobility are the predominant complaints in disease of the joints The pain may vary from mild discomfort on use to agony present even with the part at rest and making all motion impossible Stiffness may be present constantly or be noticed only after a period of rest, with disappearance after "limbering up" Restriction may vary from slight interference with function to complete fixation. Sometimes the patient will seek advice because of some more objective manifestation, such as swelling, redness, tenderness, distortion, or coldness or excessive sweating of an extremity reflecting a vasomotor component

At times the above indications will be less prominent than systemic symptoms such as fatigue, anorexia, weight loss, fever, or other variant attributable to the underlying disease as, for example, headache due to arthritis of the cervical spine, or the lacrimation and photophobia of uveitis complicating rheumatoid arthritis.

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Tests for Motions

Tests for motion must be performed with the utmost gentleness, pressure or any movement of a badly inflamed joint will cause excruciating pain. Both active and passive motion should be tested, and the corresponding joints always should be compared. In a routine examination, estimation of the degree of restriction or hypo- or hypermobility on the basis of experience may be sufficient; where indicated, more exact measurements can be taken with a goniometer In judging the degree of impairment, allowance must be made for such factors as age and habits of exercise

The methods of measuring joint function as proposed by Cave and Roberts[1] and in use at the Massachusetts General Hospital as well as many other clinics are as follows:

General Principles.

1. All motions should be measured by degrees from a neutral point of zero
2. The neutral point from which the motion is measured must be defined.
3. It is always worth while to mention the comparative motions in the joint of the opposite limb.
4. Angles should be measured with a goniometer or protractor
5. Motions of joints above and below the affected part should be measured.

Spine. Neutral position cannot be defined

1. Forward bending—this motion cannot be measured accurately in degrees, but should be compared with the probable normal for the age of the patient It should be noted whether the lumbar spine flattens or reverses itself Motions should be carried out in both sitting and standing positions.
2. Extension—it should be noted to what degree the dorsal and lumbar curves change.
3. Lateral bending—right and left
4. Rotation with pelvis fixed—right and left, comparing angle made by the shoulders with pelvis

Neck. Neutral position is with head up and chin in

1. Rotation—right and left
2. Flexion
3. Extension
4. Lateral bending—right and left

Shoulder Neutral position is arm to side, elbow flexed to 90 degrees, forearm pointing forward.

1. Flexion
2. Extension
3. Abduction—maximum 90 degrees.
4. Rotation in abduction.

[1] Cave, E F, and Roberts, S M A Method for Measuring and Recording Joint Function J Bone & Joint Surg, 18, 455 (April) 1936 Verbatim description of the tests and illustrations reproduced with permission of Dr Cave and *Journal of Bone and Joint Surgery*

fracture, impaired bone growth as in poliomyelitis, epiphyseal injury or disease, nutritional deficiency especially rickets, and bowing, as in Paget's disease. Moderate or marked shortening is obvious, lesser degrees must be sought by careful measurements Arm length is measured from the tip of the acromium to the ulnar styloid with the patient standing and his arm at his side Lower extremity measurement is described in Chapter 34. In low chronic backache, measurement may be indicated to exclude shortening of one lower extremity as the cause; inequality of limb length, by creating pelvic tilt and secondary spinal curvature, will eventually give rise to the picture of back strain.

Crepitus. Bony, cartilaginous or fibrous overgrowth, destruction of articular surfaces, and free bodies in the joint are the common causes of crepitus It can be felt by resting one hand on the joint while the part is passively put through its range of motion Pronounced crepitus is audible (*creaking*)

Restricted Joint Motion. The tests for joint mobility are described below Restricted motion is due to one or more of the following.

1. Pain

2 Muscle spasm. Active and passive movements are attended by pain and resistance, usually beginning with onset of motion and gradually increasing until movement is stopped at some point vaguely determined by one's strength and hardness of heart, and the patient's threshold of pain On palpation, the involved muscles are tender and feel tense or rigid. Spasm of the erector spinae muscles is often apparent at a glance the patient stands rigidly and all motions of his back are guarded Spasm of muscles around the hip joint is of especial importance There are two types· that due to irritation of the psoas muscle alone and that in which all the muscles are involved In pure *psoas* spasm, the thigh is partially flexed on the trunk and extension is restricted; other motions are not hampered. A slight degree of trouble with this muscle is indicated solely by limitation of passive hyperextension, best demonstrated with the patient prone In *generalized* spasm of the hip muscles, movement in all directions is restricted

3 Muscular contracture. This results from tonic spasm of one group of muscles, or loss of muscular equilibrium due to paralysis of its opposing group The muscle is usually non-tender, the affected part is held in an abnormal position, and mobility is decidedly impaired

4. Inflammation, thickening or adhesions in the capsular or periarticular structures Movement is at first painful and restricted but becomes less so after activity. The regional muscles may or may not be tense

5. Effusion Although a large effusion may limit function, restriction is more likely due to accompanying muscle spasm and capsular changes.

6 Bony or cartilaginous overgrowth Motion is usually free up to a point, at which it is suddenly and completely, often painlessly, arrested Rarely, a gouty tophus will create this variant.

7. Bony ankylosis. There is no mobility whatsoever.

Hypermobility. As a result of destruction of joint surfaces or relaxation of supporting structures, the range of motion is greater than normal.

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Tests for motion must be performed with the utmost gentleness, pressure or any movement of a badly inflamed joint will cause excruciating pain. Both active and passive motion should be tested, and the corresponding joints always should be compared. In a routine examination, estimation of the degree of restriction or hypo- or hypermobility on the basis of experience may be sufficient, where indicated, more exact measurements can be taken with a goniometer. In judging the degree of impairment, allowance must be made for such factors as age and habits of exercise

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2 Flexion
3 Extension
4 Lateral bending—right and left

Shoulder Neutral position is arm to side, elbow flexed to 90 degrees, forearm pointing forward

1 Flexion
2 Extension
3 Abduction—maximum 90 degrees
4 Rotation in abduction

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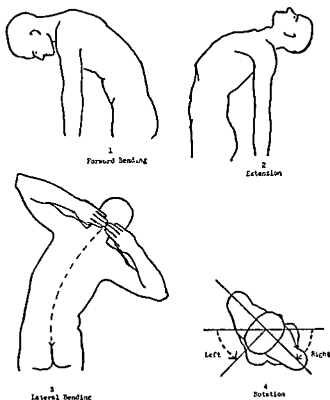


Fig 35 2 Motions of spine

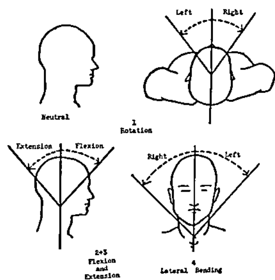


Fig 35 3 Motions of neck

5 Rotation in neutral (arm behind back to test extreme internal rotation—compared with opposite side).

6. Elevation—compared with opposite side and measured in number of degrees. (This is shoulder girdle motion as compared with items 1 to 5, which are true humeroscapular motions.)

Elbow. Neutral position is with forearm in extension.

1. Flexion—measured from complete extension, the neutral point.

2. Hyperextension—measured in degrees as compared with the opposite elbow.

3. Supination from a neutral point—which is midposition between pronation and supination.

4. Pronation—elbow must be fixed at side in 90 degrees of flexion

5 When there is loss of complete extension, this loss should be recorded in degrees of permanent flexion

Wrist. Neutral position is with hand in line with forearm with palm down.

1. Dorsiflexion (extension).

2. Palmar flexion.

3. Ulnar deviation.

4. Radial deviation

5. Pronation }
6. Supination } to be noted as described under *Elbow*

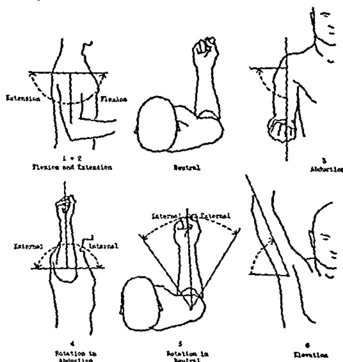


Fig 35 4 Motions of shoulder.

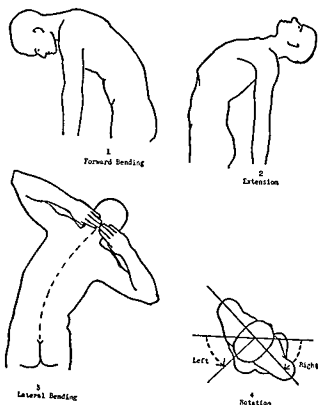


Fig 35 2 Motions of spine

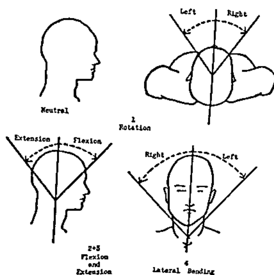


Fig 35 3 Motions of neck

1. Flexion measured with the knee bent Opposite thigh must remain in neutral.

1A To test permanent flexion, the opposite thigh must be flexed, so as to flatten the lumbar spine and to fix the pelvis

1, but with the patient ly-

an angle of 90 degrees

n angle of 90 degrees with

a line joining the anterior-superior spines.

4. Adduction—the same.

5. Rotation (external and internal) in extension Measurement should be made with patient prone and knee flexed to 90 degrees

6. Rotation (external and internal) in flexion Measurement should be made with patient on back with knee and thigh flexed to 90 degrees

Knee. Neutral position is complete extension

1 Flexion—measured in degrees from complete extension

2 Hyperextension

3. Anteroposterior stability should be tested with the knee in 90 degrees of flexion. Lateral stability should be tested with the knee in complete extension

4. When there is loss of complete extension, it should be recorded in degrees of permanent flexion

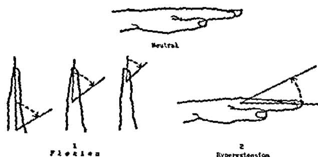


Fig 35 7 Motions of fingers

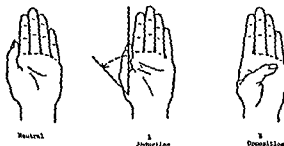


Fig. 35 8. Motions of thumb.

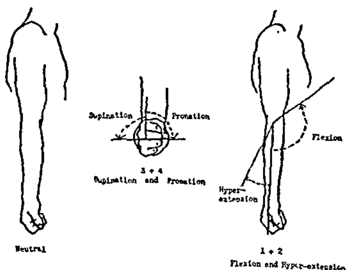


FIG 35 5 Motions of elbow

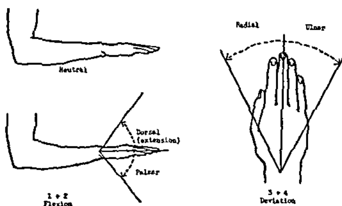


FIG 35 6 Motions of wrist

Fingers. Neutral position is with fingers in extension

1. All motions are in flexion, either in the metacarpophalangeal or interphalangeal joints.

2 Hyperextension should be noted if present.

3. Test should be made for increased lateral mobility.

Thumb. Neutral position is with thumb alongside the forefinger and extended.

1. Abduction—measured by the angle that the thumb makes with the forefinger.

2. Flexion—measured the same as for the fingers

3. Opposition—cannot be measured in degrees, it should be noted how far thumb comes across the palm.

Hip. Neutral position is with hip in extension, patella pointing upward

1. Flexion measured with the knee bent. Opposite thigh must remain in neutral.

1A. To test permanent flexion, the opposite thigh must be flexed, so as to flatten the lumbar spine and to fix the pelvis

2. Hyperextension—neutral, the same as for flexion, but with the patient lying prone with opposite thigh over the end of table at an angle of 90 degrees

3. Abduction—measured from a line which forms an angle of 90 degrees with a line joining the anterior-superior spines.

4. Adduction—the same

5. Rotation (external and internal) in extension Measurement should be made with patient prone and knee flexed to 90 degrees

6. Rotation (external and internal) in flexion Measurement should be made with patient on back with knee and thigh flexed to 90 degrees

Knee. Neutral position is complete extension

1. Flexion—measured in degrees from complete extension

2 Hyperextension.

3. Anteroposterior stability should be tested with the knee in 90 degrees of flexion. Lateral stability should be tested with the knee in complete extension

4 When there is loss of complete extension, it should be recorded in degrees of permanent flexion.

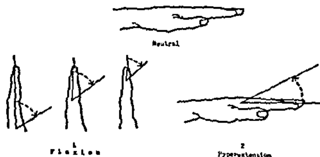


FIG 35 7 Motions of fingers



FIG. 35 8 Motions of thumb.

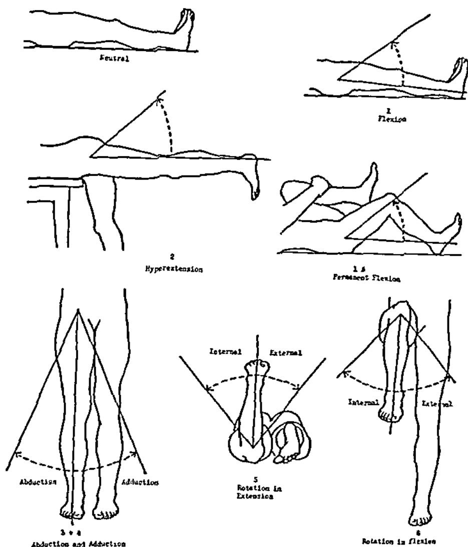


FIG 359 Motions of hip

Ankle. Neutral position is with the outer border of the foot at 90 degrees with the leg and in neutral as regards inversion and eversion

1. Dorsiflexion should be tested with the foot in inversion Measurements should be compared with knee flexed and with knee in extension, to rule out tight calf muscles.

2. Plantar flexion.

Foot. Neutral position cannot be defined.

1. Subastragalar motion is inversion and eversion.

2 Mediotarsal joints (forefoot adduction and abduction) tested passively with the os calcis held in neutral.

3. Metatarsophalangeal joints—particularly important in the great toe.

4 Interphalangeal joints—tested in flexion and extension, and for increased lat

and in which direction there is

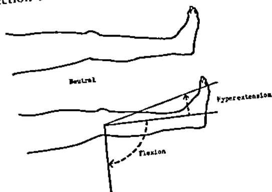


FIG 35 10 Motions of knee



FIG 35 11 Motions of ankle

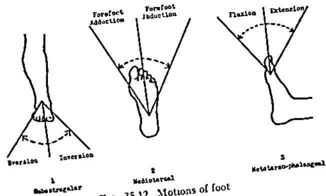


FIG 35 12 Motions of foot

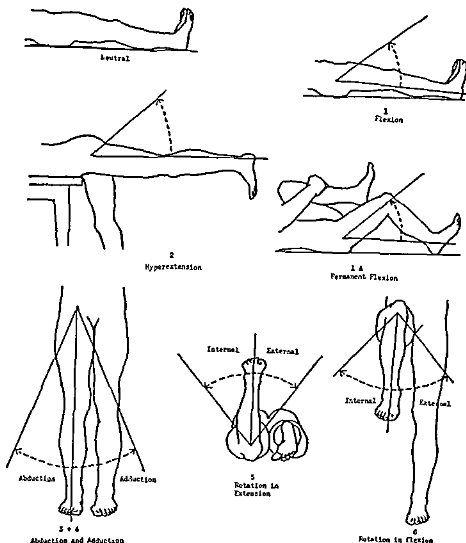


FIG 35 9 Motions of hip

Ankle. Neutral position is with the outer border of the foot at 90 degrees with the leg and in neutral as regards inversion and eversion

1. Dorsiflexion should be tested with the foot in inversion Measurements should be compared with knee flexed and with knee in extension, to rule out tight calf muscles.

2. Plantar flexion

Foot. Neutral position cannot be defined.

1. Subastragalar motion is inversion and eversion

2. Mediotarsal joints (forefoot adduction and abduction) tested passively with the os calcis held in neutral

3. Metatarsophalangeal joints—particularly important in the great toe.

Erythema nodosum
Hemophilia
Hysteria
Ochronosis
Osteochondritis dissecans
Osteochondromatosis
Periarteritis nodosa
Psoriasis
Purpura, various types
Raynaud's disease
Reiter's disease
Scleroderma
Serum sickness

In the following pages we omit discussion of some of the less common disturbances noted above but include description of certain other diseases which can be confused with arthritis For convenience, we deviate at times from the order of classification

Arthritis Due to Infection

Acute Pyogenic Arthritis. This may be caused by any of the pyogenic organisms, such as Streptococcus, Staphylococcus, Pneumococcus, Gonococcus, Meningococcus or colon Bacillus. Severe cases have become much less common since the introduction of antibiotic therapy The degree to which the joints are involved is extremely variable, ranging from the case of extensive joint disease with or without pronounced systemic response to that in which systemic and other manifestations of the underlying infection overshadow joint changes. Between these extremes one can encounter any combination. For example, arthritis in severe Pneumococcus pneumonia or acute meningococcic infection appears only as one of the manifestations of overwhelming disease, whereas in chronic low-grade meningococcic infection, the joint changes may predominate with only mild systemic symptoms In the mild to moderate case, one will find some degree of fever and associated malaise, migratory aches and pains or, for a day or two, migratory polyarthritis, usually affecting the large joints During this phase the clinical picture suggests rheumatic fever As a rule, most of the joints initially involved will become symptomless within a few days but frequently one or more—usually the larger—will continue to cause trouble, showing pain, redness, swelling, and increased surface temperature Recovery within days or weeks with little or no residual disability is the rule.

In the more serious case, the inflammatory changes in one or more joints may create purulent effusion (*purulent* or *septic arthritis*) Pain, swelling, redness, and heat are much more severe Fluctuation due to presence of pus can be demonstrated If appropriate treatment is not initiated early, joint destruction will progress rapidly and probably end in permanent disability due to subluxation, or fibrous or bony ankylosis

X-ray is negative in the mild or early severe case except for soft tissue swelling, perhaps widening of the joint space by effusion, and occasionally, moderate de-

X-Ray Examination

X-ray may detect or confirm the following:

1. Soft tissue swelling due to periarticular thickening or increased fluid in the joint
2. Bony overgrowths—their *shape, extent, and position*
3. Decalcification or sclerosis of bone near a joint
4. Necrosis, tumor, or cyst near joint line.
5. Narrowing of joint space due to destruction of cartilage
6. Subluxation and dislocation
7. Calcified body within the joint

Other Investigations

In addition to the procedure just described, survey of the patient must include a search for other manifestations of the disease responsible for the joint changes, such as rheumatoid arthritis, gout, gonorrhea, brucellosis, tuberculosis, non-tuberculous pulmonary disease, or tabes dorsalis Tuberculin reaction, serologic test for syphilis, blood sedimentation rate, uric acid, calcium, phosphorus and phosphatase determinations, examination of the spinal fluid, and other special tests are often required

RHEUMATIC DISEASES

Classification

The following classification is a summary of that recommended by the American Rheumatism Association [2]

Arthritis due to infection
Arthritis due to rheumatic fever
Rheumatoid arthritis (Proliferative arthritis, atrophic arthritis, chronic infectious arthritis)
Arthritis due to trauma
Neurogenic arthropathy
Gout
Degenerative joint disease (osteoarthritis, hypertrophic arthritis, degenerative arthritis, chronic senescent arthritis)
New growths of joints
Intermittent hydrarthrosis
Fibrositis
Myositis, bursitis, neuritis, neuralgia

Diseases in which Arthritis, Arthropathy, or Arthralgia is Frequently Associated

Acromegaly
Acute disseminated lupus erythematosus
Cyst of meniscus of knee
Dermatomyositis
Drug intoxication
Erythema multiforme exudativum

[2] Committee of the American Rheumatism Association Primer on the Rheumatic Diseases, J A M A 152, 323, 152, 405, 152, 522 (May 23, May 30, June 6) 1953

Erythema nodosum
Hemophilia
Hysteria
Ochronosis
Osteochondritis dissecans
Osteochondromatosis
Periarteritis nodosa
Psoriasis
Purpura, various types
Raynaud's disease
Reiter's disease
Scleroderma
Serum sickness

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Arthritis Due to Infection

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X-ray is negative in the mild or early severe case except for soft tissue swelling, perhaps widening of the joint space by effusion, and occasionally, moderate de-

calcification. In the long-standing severe case, one will find marked destruction of joint surfaces, subchondral bone necrosis, marked spotty atrophy, and narrowing of joint space. Months later, recalcification and ankylosis may be apparent.

Tuberculous Arthritis. Usually beginning in childhood or adolescence, this is most common in the spine, hip, or knee, in the order named. Onset is insidious. Night cries preceding any local manifestation may be the first hint of trouble. With time, one finds disability first noted as limp, spasm, swelling, limited active and passive motion, muscular atrophy, deformity, and, in the lower extremity, shortening. Occasionally subluxation and sinuses develop but thanks to earlier diagnosis and improved methods of treatment, these are less common than formerly. In the long-standing case, a sinus may become secondarily infected and eventually lead to amyloid disease. Early in the course, x-ray is negative except for increased density representing soft tissue swelling. Later, progressive destruction of articular surfaces, and narrowing and irregularity of joint spaces are seen, evidence of bone repair is minimal or absent. Sometimes the x-ray picture is indistinguishable from that of severe acute pyogenic arthritis but the two can usually be clinically distinguished.

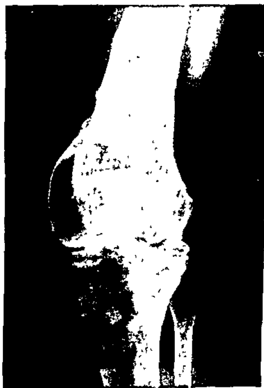


Fig. 35 13 Tuberculosis of knee, advanced. Extensive destruction of articular surfaces with narrowing and partial obliteration of joint space. Patches of diminished density due to bone destruction also evident.

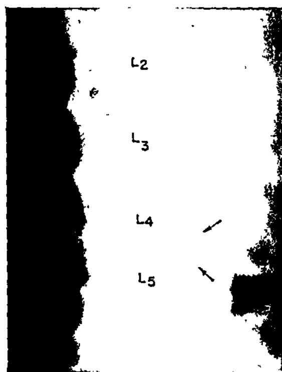


FIG 35 14 Tuberculosis of lumbar spine in a man age 49 with known pulmonary tuberculosis for 3 years Lateral film taken shortly after onset of low back pain Intervertebral space between L4 and L5 narrowed Articular surface of L4 shows irregularity and some areas of destruction, of L5, complete disintegration Partial bone destruction of L5 indicated by areas of diminished density Secondary new bone formation, more pronounced than usually occurs in tuberculosis, evident anteriorly (arrows) (Courtesy Middlesex County Sanatorium, Waltham, Massachusetts)

TUBERCULOSIS OF SPINE (POTT'S DISEASE) This shows, in addition to the usual indications of joint tuberculosis, posterior knuckling or curving of the spine (*kyphosis*) due to destruction of vertebral bodies The lesion is frequently accompanied by *cold abscess* formation Detritus originating in the cervical spine is most likely to collect in the retropharyngeal space, causing dyspnea and dysphagia, or point superficially on either side of the neck In disease of the thoracic spine, the abscess may remain small and absorb; if larger, it can extend into the posterior mediastinum, penetrate the pleura, giving rise to tuberculous empyema, point superficially on the thoracic wall, or gravitate downward along the spine and pursue the course followed by discharge from a lumbar abscess. Pus from the lumbar spine is most likely to follow the course of the psoas muscle (*psoas abscess*), giving rise first to psoas spasm, and later pointing in the thigh, groin, or buttock. Occasionally in the untreated or long-standing case, paralysis or other neurologic complication will result from cord compression; this is most likely to occur when the thoracic spine is diseased.

Syphilitic Arthritis. CONGENITAL In *infancy*, swelling at the epiphyseal junc-

tions, especially the distal ends of the bones of the lower extremities, occurs as a result of osteochondritis. The mild case shows moderate effusion into the joint, due to synovitis; the more severe case may proceed to destruction of joint surfaces X-rays show thickening and irregularity of the epiphyseal line, or, when the process has advanced, degeneration and a line of necrosis between epiphysis and diaphysis.

In *childhood or later*, one finds thickening and roughening of the long bone shafts, resulting from earlier osteitis or periosteitis These changes are particularly easy to discover in the tibia (*saber-shin*) or in the clavicle Painless bilateral effusion due to syphilitic tenosynovitis is not uncommon (*Clutton's joints* or *tenosynovitis syphilitica*). Although any joint can be involved, the knees are the most frequent sites Other stigmata of congenital syphilis, such as interstitial keratitis and Hutchinsonian teeth are usually, but not necessarily, evident The course is chronic with minimal active inflammation or functional impairment. Spontaneous recovery without residual joint change is the rule; response to antisyphilitic treatment is slight This variant may be confused with early tuberculous arthritis. Rarely, dactylitis (*see* Chap 6) is a reflection of syphilis

Acquired During the *early* stage, in addition to myalgia which is common, the patient may complain of pain, tenderness, or other symptoms suggesting arthritis in one or more joints, a mistaken diagnosis of rheumatic fever or rheumatoid arthritis may be the result. The pain is worse at night or following immobility, and relieved by use Effusion into one or more joints, usually the knees, elbows or ankles, sometimes occurs. These variants are transient X-rays show no important changes

In *late* syphilis, a gummatous process originating in adjacent bone may extend into a joint The knees and elbows are the usual sites, involvement is often symmetrical One finds soft tissue swelling with shiny, tense skin, pain is absent or slight Mobility is impaired X-ray shows soft tissue swelling, destructive or proliferative changes in bone, and perhaps a fine lace-like reaction in the periosteum, joint effusion may be evident *Charcot joint*, which occurs in tabes dorsalis, is described under neurogenic arthropathy

Miscellaneous Infections. Varying degrees of pain, stiffness, swelling, and other indications of articular or periarticular inflammation are sometimes encountered in mononucleosis, subacute bacterial endocarditis, brucellosis, bacillary dysentery, lymphogranuloma venereum, and certain other less common infectious diseases As a rule, the joint manifestations are transient, permanent damage is rare. At times, arthralgia or low-grade arthritis of one or more joints is a reflection of a remote and perhaps quiescent focus of infection, especially in the tonsils, paranasal sinuses, or prostate. Cure can be effected by proper treatment of the primary source However, it is also true that a case initially regarded as belonging to this group often will not be relieved by treatment of the suspected focus and may eventually turn out to be one of rheumatoid arthritis.

Arthritis Due to Rheumatic Fever

Rheumatic fever and its complications are described in Chapter 16.

Rheumatoid Arthritis

This is a chronic, progressive, polyarticular disease of unknown cause developing at any age and affecting about the same percentage of all age groups beyond the second decade. Incidence is higher in females The disease may begin acutely or insidiously, start in one joint or in several small or large ones, and occasionally, at onset, resemble the picture of acute infectious arthritis The most commonly involved joints are knees, proximal interphalangeals of the hands, wrists, metacarpophalangeals of hands and feet, hips, sternoclaviculars, temporomandibulars, ankles, and spine When fully developed, the process is symmetrical, corresponding joints being equally affected The majority of cases show spontaneous arrest but retain residual joint changes; a small percentage recover spontaneously, another small percentage pursue a relentless course ending in total physical incapacity Exacerbations and remissions are the rule, the process may become arrested or quiescent at any time, even in the most severe case, or the quiescent case may, at any time, become mildly to severely reactivated

Local Changes. Pain, tenderness, subjective stiffness, swelling with or without effusion, and, rarely, redness and heat, are the initial local manifestations. With progression of the disease, one finds muscle spasm, limitation of motion, greater stiffness, weakness, muscular atrophy, and eventually deformity, often with varying degrees of ankylosis or subluxation Pain and other indications of acute inflammation are dependent at any time on the degree of activity The regional muscles show atrophy greater than can be explained by disuse alone Periarticular swelling in the chronic stage is less pronounced than when the process is acute but the joints are likely to appear large because of soft tissue atrophy In the fingers, the swelling is fusiform and, in contrast to degenerative joint disease, the terminal joints are rarely involved. The overlying skin shows changes indicated below In the advanced case, the hands and fingers are fixed in various combinations of flexion and extension, usually with ulnar deviation of the phalanges In the feet, one finds plantar flexion, depression of metatarsal heads, and lateral deviation of the toes The hips are fixed in flexion and adduction; the knees show flexion deformity, sometimes with posterior subluxation of the tibia on the femur, the shoulders, adduction and internal rotation, the elbows and wrists partial flexion Partial or complete fixation of the jaw may hamper opening of the mouth Changes in the spine are described below.

Constitutional Disturbances. Fatigue, lassitude, vasomotor variants, and paresthesias accompany the joint changes and at times precede them. In the advanced case, one finds pronounced general weakness, poor nutrition, and sometimes actual cachexia. Low-grade fever is the rule, although in the chronic case temperature may be normal. During an acute phase, fever reaching as high as



FIG 35 15 Rheumatoid arthritis, duration 2 years, in a woman age 45 Fusiform swelling of proximal interphalangeal joints Slight flexion deformity of left fourth and right fifth fingers, ulnar deviation of left fifth finger Circumscribed prominence on each wrist represents a collection of synovial fluid originating in carpal joints In contrast to degenerative joint disease, most terminal joints appear normal (Courtesy Dr Leonard W Cronkhite, Jr)

103°–104° may persist for some time In a small percentage of cases, the disease begins with high fever and acute polyarthritis simulating acute rheumatic infection. The pulse is likely to be rapid, even in the afebrile patient Although the blood picture is variable, mild-to-moderate hypochromic anemia is the rule Leukocyte count may be elevated in the more acute phases, in the milder ones, it is normal or low, with a relative lymphocytosis Sedimentation rate is almost always elevated It is an important indication of activity, and valuable in differentiating rheumatoid from degenerative arthritis.

Cutaneous Changes. Over the extremities, the skin is atrophic, shiny, and usually cold and moist Sometimes it becomes cyanotic, Raynaud's-like episodes may occur Marked redness of the thenar and hypothenar eminences is not uncommon Light-to-deep-brown pigmentation is possible, chiefly on the extremities and face, it can be diffuse or patchy and is likely to be interspersed with patches of vitiligo Psoriasis is found in a small number of cases, if it involves the nails the terminal phalangeal joints almost always show arthritic changes

Subcutaneous Nodules. These are likely to be found on the extensor aspects of the joints, most commonly just below the olecranon process and, in bedridden patients, along the spine, on the posterior aspects of the scalp, and in the sacroiliac regions. Varying in size from 1–2 mm to 1–2 cm, these nodules are usually freely movable beneath the skin, but may be attached to the tendons. They are rarely painful except when situated on pressure points

Other Changes. Enlargement of the spleen is noted in 10–20 per cent of cases,



FIG 35.16. Rheumatoid arthritis Metacarpophalangeal and proximal interphalangeal joints show varying degrees of narrowing of spaces, irregularity and destruction of surfaces, destruction of subchondral bone, and deformity. Distal interphalangeal joints narrowed but show no destruction Carpal joints narrowed and show some bony change Diffuse decalcification, more pronounced adjacent to joint surfaces, is also evident

usually children Lymphnodes may be enlarged, especially those which receive drainage from the regions of diseased joints In any large series, rheumatic heart disease is found in about 3 per cent of cases Pericarditis and pleuritis occasionally occur, especially in children

X-ray Findings. In the earliest stages, films may be negative or show soft tissue swelling, and decalcification Somewhat later, bone atrophy is observed As the disease advances, one will find narrowing of the joint spaces due to cartilage destruction, subchondral areas of bone destruction, deformity, and, when damage is extreme, subluxation and bony ankylosis.

Variants of Rheumatoid Arthritis

The following disturbances are generally regarded in most clinics as probable forms of rheumatoid arthritis, although the relationship has yet to be clearly established.

Rheumatoid Spondylitis. Although changes in the spine, lumbosacral, and sacroiliac joints often occur as a feature of polyarticular rheumatoid arthritis, they sometimes develop independently (*rheumatoid spondylitis, Marie-Strumpell spondylitis, von Bechterew spondylitis*) Some authorities regard this process as a separate entity but others, including the Arthritis Service of the Massachusetts General Hospital, look upon it as a form of rheumatoid arthritis In contrast to the latter, its incidence is decidedly higher in males. The earliest indications are usually low-back pain, limitation of motion on forward bending, soreness in the buttocks, and sometimes pain referred down the sciatic nerves The localization, and absence of x-ray findings at this stage often lead to a mistaken diagnosis of low-back strain. With time the process advances to include more and more of the spine, creating wider distribution of pain and greater restriction of back and head movements in all directions Spasm and tenderness of the erector spinae muscles are pronounced Ultimately the back becomes completely fixed with lumbar curve flattened, thoracic curve exaggerated, and neck in partial flexion (*poker spine*) Ankylosis of the costovertebral joints fixes the thorax so that there is little or no respiratory expansion, and breathing is of the abdominal type. Root pain may cause confusion with a visceral disturb-



Fig. 35 17 Rheumatoid spondylitis, early Increased density of bone adjacent to both sacroiliac joints with beginning obliteration of joint spaces.



Fig 35 18 Rheumatoid spondylitis, advanced (bamboo spine) Smoothly-contoured bridging of intervertebral spaces by calcification of longitudinal ligaments Some decalcification of vertebral bodies Obliteration of sacroiliac joint space evident on left, similar process present on right lost in reproduction

ance such as angina pectoris or cholelithiasis As a rule, the disease is slowly but definitely progressive Exacerbations and remissions, although possible, are not as striking as in generalized rheumatoid arthritis, and constitutional manifestations are not as pronounced X-ray first shows haziness with later narrowing or obliteration of the sacroiliac joints and (best seen in oblique views) of the apophyseal joints. In the advanced case, a striking picture (*bamboo spine*) is created by decalcification of the vertebral bodies and diffuse calcification of the lateral spinal ligaments.

In about one-quarter of cases, some changes characteristic of rheumatoid arthritis will develop in the peripheral joints, especially hips, shoulders, or knees, but the process in the spine is decidedly predominant

Still's Disease. This term is applied to rheumatoid arthritis in children. The picture is similar to that seen in adults but one may find, in addition, striking enlargement of lymphnodes and spleen, and in the severe case, pericarditis, pleuritis, or cardiac hypertrophy without evidence of valvular lesions Systemic response is sometimes severe, with fever, anemia, leukocytosis, rapid wasting,

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senting bizarre or atypical forms of rheumatoid arthritis. They are characterized by intermittent episodes of joint effusion sometimes occurring at regular intervals, and usually involving the knees Occasionally, mild trauma without any other obvious predisposing factor causes the joint to become swollen and show signs of effusion More often no such inciting factor is found Pain and discomfort are not severe, but motion may be impaired by the effusion. Signs of inflammation, such as redness and heat, are absent Constitutional symptoms are minimal or absent. Attacks may recur for years without leaving any residual changes

Palindromic Rheumatism. This rare syndrome is marked by multiple, transient episodes of pain, swelling, redness, and tenderness involving a joint, the periarticular structures alone, or sometimes the skin and subcutaneous tissue at some distance from a joint As a rule, only one area is involved at a time. The hands, feet, and knees are the most likely sites Attacks begin abruptly and usually spontaneously subside within 1–3 days, intervals between them are extremely variable, ranging from a few hours to a year or more Constitutional symptoms, abnormal laboratory findings, and joint changes by x-ray are absent or minimal.

Occasionally attacks of this nature are encountered in a patient with well-established rheumatoid arthritis, or in one who develops this disease later.

Reiter's Syndrome. This term refers to a relatively small group of cases marked by a triad of urethritis, conjunctivitis and arthritis, which usually appear in that order within a period of days to weeks Although the cause has not been determined, it is well established that the Gonococcus is not blameable The urethritis is of relatively short duration, although the discharge can vary from serous to purulent, it is less profuse and of shorter duration than in gonorrhea The conjunctivitis, usually bilateral, may be minimal to severe The arthritis, usually polyarticular and more likely to involve weight-bearing joints, simulates low-grade infectious arthritis but is of longer duration, effusion, when present, is less purulent

Other manifestations sometimes encountered are cystitis, uveitis, diarrhea, especially during the early stages, and characteristic skin lesions which are marked by vesiculation and hyperkeratosis and are most likely to involve the penis, mouth, hands, and feet Constitutional symptoms are minimal to quite severe The disease is long-standing and, as a rule, subject to remissions and exacerbations

Joint Changes in Ulcerative Colitis. In idiopathic ulcerative colitis, especially when the disease is long-standing, joint changes indistinguishable from those of rheumatoid arthritis develop in a certain number of cases If the colitis is alleviated by medical or surgical means, they will often subside, but in some patients chronic, deforming arthritis persists.

Arthritis Due to Trauma

Acute. Usually resulting from a direct blow or sudden strain, this is characterized by pain, swelling with or without effusion, local tenderness, spasm, re-

and pronounced changes such as demineralization of bone, contractures and deformities. Initially, the disease may be confused with rheumatic fever but later permanent joint changes clarify the diagnosis. By no means do all cases of rheumatoid arthritis in children show this picture; some are relatively mild and develop few sequelae.

Felty's Syndrome. Also called *rheumatoid arthritis with hypersplenism*, this includes a small group of patients with chronic arthritis of the rheumatoid type accompanied by leukopenia and moderate to great enlargement of the spleen. At the Massachusetts General Hospital, it is thought that such cases actually are true rheumatoid arthritis and that the splenic enlargement and associated leukopenia are a reflection of an unrelated disturbance.

Psoriatic Arthritis. Psoriasis occurs in approximately 3 per cent of patients with rheumatoid arthritis. In a small number of cases, in contrast to the usual form of the latter, the terminal joints of the fingers are involved. The nails show the characteristic psoriatic scarring and pitting. Some observers believe that this type may represent a separate entity; others that it is a variant of rheumatoid arthritis.

Possible Variants of Rheumatoid Arthritis

Authorities differ widely as to whether or not the following disturbances are related to rheumatoid arthritis. Although convincing proof is lacking, there is enough suggestive evidence of relationship to justify our including them here.

Fibrositis. Inflammatory changes appear to be confined to the muscles, the periarticular structures, or both; there are no clinical or pathologic changes within the joints. Since, in the early stages, the disease cannot be distinguished from beginning arthritis, the diagnosis of fibrositis is justified only if, with time, no evidence of joint involvement appears. Furthermore, although the patient may complain of a certain degree of general weakness, other systemic variants encountered in rheumatoid arthritis such as anorexia, loss of weight, anemia, debility, leukocytosis, and increased sedimentation rate, are not the rule.

When the *muscles* are primarily involved, the symptoms are aching, subjective stiffness or soreness in the muscles; sometimes an acute episode with tenderness and spasm occurs. Discomfort is usually worse after rest and becomes less after "limbering up". Parts especially likely to be involved are the cervical, dorsal, or lumbo-sacral regions of the back, and the shoulder girdle. Rarely, subcutaneous thickening or definite nodules are found overlying the muscles. Such disorders as recurrent stiff neck and lumbago probably are the result of intramuscular fibrositis. Involvement of *periarticular structures* is indicated by pain and stiffness with perhaps tenderness referred to the joints. The muscles may or may not be affected. Swelling, redness, heat, and other signs of joint inflammation are lacking. Although contractures sometimes occur with associated muscle spasm, deformities and other late signs of joint disease do not develop. X-rays show no changes.

Intermittent Hydrarthrosis. By the Arthritis Service at the Massachusetts General Hospital, most cases of intermittent hydrarthrosis are regarded as repre-

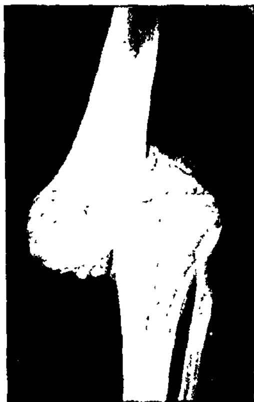


FIG 35 19 Charcot knee in a man age 59 with long standing tabes dorsalis Destruction of joint surfaces, disintegration of underlying bone, and subluxation Numerous bone fragments evident in joint space.

phalangeal of the large toe—the traditional site of the process—is affected, or because the patient does not necessarily live intemperately. There are no established grounds for blaming indulgence in rich food and alcoholic drinks for the disease itself, although temporary excess is accepted as one of the factors conducive to an acute episode Other precipitating factors are trauma, surgical operation, systemic infection, excessive intake of liver extract or thiamin, high fat or purine diet, mercurial diuretic, fatigue, and emotional stress.

Rarely, gout is encountered as a result of increased nucleoprotein catabolism in certain blood dyscrasias such as polycythemia, hemolytic anemia, myeloid metaplasia and chronic leukemia.

Acute Stage. Ordinarily, the initial attack occurs in mid-life or later, but sometimes as early as the second or third decade. At first there will be intervals of years between episodes but, with time, periods of freedom are likely to become shorter even to the point where the patient has several attacks a year

The onset of acute gouty arthritis is often dramatic, severe pain in one or more joints appearing suddenly, most often during the night For a day or so beforehand, the patient may have been aware of a vague sense of ill-being or gastro-

striction of motion, and sometimes discoloration. X-ray examination is usually negative, except for the increased density of effusion or soft tissue swelling As a rule, recovery follows spontaneously or after proper treatment.

Chronic. The cause is improperly treated acute trauma, repeated mild trauma, or prolonged strain. The picture varies with the degree of injury, and the use to which the joint is subjected. With the part at rest there may be no symptoms, but activity will provoke pain, tenderness, and perhaps swelling and impaired motion These may disappear with rest only to reappear when the joint is again put to use. In the long-standing case, permanent changes in the joint will result in persistence of the signs and symptoms indicated, irrespective of activity This stage can be regarded as a form of degenerative joint disease X-ray will be negative or, in the well-established case, show degenerative changes, such as irregularity and narrowing of joint spaces, and calcium deposits in the ligamentous attachments

Neurogenic Arthropathy

In tabes dorsalis, syringomyelia, peripheral nerve injury, and other neurologic disturbances with impaired sensation, especially proprioceptive sensation, joint changes are not uncommon They are presumably due to repeated trauma not apparent to the patient because of the hypesthesia. Strictly speaking, the term *Charcot joint* applies only to the process when it occurs in tabes dorsalis, but actually it is generally used to include comparable changes occurring in the other disturbances mentioned In tabes dorsalis, the knees, less often the ankles or feet, are the usual sites, in syringomyelia, the joints of the upper extremities Painless enlargement, due largely to serous effusion, is often the first indication Gradual destruction of joint surfaces and ends of the bones, proliferative changes, and relaxation of ligaments eventually result in deformity, hypermobility, dislocation and subluxation; pain is absent or minimal X-ray shows narrowing of the joint spaces, destruction of cartilage, and frequently irregular destruction and eburnation of adjacent bone with perhaps calcification representing fragments of bone in the soft tissues

Following a cerebro-vascular accident, the patient may develop pain, swelling, and stiffness of one or more joints of an affected extremity, presumably from a combination of disuse, vasomotor impairment, and perhaps abnormal pull of spastic muscles.

Gout

In patients with gout, there is an alteration of purine metabolism, the cause of which is not known. That it is probably an inborn disease of metabolism is attested to by the fact that perhaps as many as 75 per cent of all gouty patients give a positive family history. The disease is rare in females It is characterized by recurrent attacks of acute arthritis, between which, as a rule, the patients are free of trouble. A relatively small number through the years develop progressive deforming joint changes. More common than is generally supposed, gout often goes unrecognized or mislabeled because a joint other than the metatarso-



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phalangeal of the large toe—the traditional site of the process—is affected, or because the patient does not necessarily live intemperately There are no established grounds for blaming indulgence in rich food and alcoholic drinks for the disease itself, although temporary excess is accepted as one of the factors conducive to an acute episode. Other precipitating factors are trauma, surgical operation, systemic infection, excessive intake of liver extract or thiamin, high fat or purine diet, mercurial diuretic, fatigue, and emotional stress

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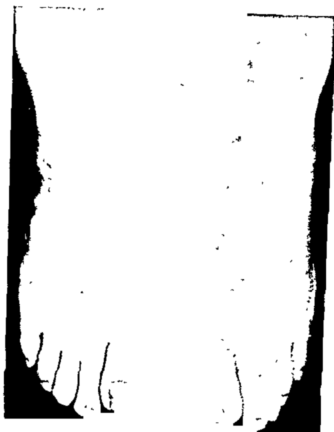


FIG 35 20 Swelling of left foot during an attack of acute gouty arthritis of tarsometatarsal joints Exquisite local pain and tenderness present

intestinal uneasiness. Any of the precipitating factors noted above may have been operative. As a rule, pain is excruciating from the start, even though the joint is kept at rest; it is aggravated by any motion, slight jarring, or the weight of the bedclothes. Within a half hour or so, the joint becomes swollen with overlying skin violaceous, tense, shiny, and hot, and regional superficial veins often distended. The swelling rapidly extends beyond the joint, for example, in a case involving the large toe, the entire foot may become edematous and perhaps red. The picture resembles and is often confused with that of cellulitis This is rarely true of other forms of arthritis. Effusion into the joint or inflammation of a neighboring bursa may develop. Although the metatarsophalangeal joint of the great toe is the classic site, it must be emphasized that gout often involves other joints singly or multiply, especially the ankles, knees, wrists, elbows, or hands and feet. Mild fever and leukocytosis are the rule during the acute phase Early attacks may be migratory and in such instances are sometimes misdiagnosed rheumatic fever. *Subcutaneous tophi, an important indication of chronic gout, are not present early in the disease* If untreated, the attack will



FIG 35 21 Chronic gout. Typical punched out areas of diminished density adjacent to joints, especially marked in second and third digits Surfaces of terminal joints of index and fifth fingers show destructive changes



FIG 35 22 Severe long standing gout Pronounced soft tissue swelling and destructive bone and joint changes of great toe First and fifth metatarsals show typical punched-out areas of bone destruction. In contrast to rheumatoid arthritis, generalized decalcification is not prominent.

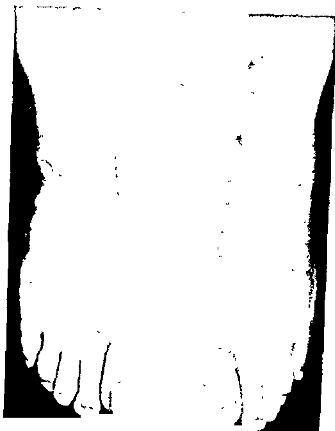


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Although the cause is unknown, it is thought to be advancing age with the wear and tear of long-continued use. This explains, perhaps, the predilection for weight-bearing joints and the relative frequency with which the trouble is encountered in the obese and in persons with mechanical skeletal defects.

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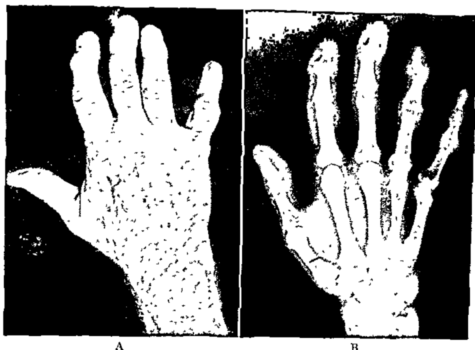


FIG 35 23 Degenerative joint disease

A Nodular swellings (Heberden's nodes) and deformities of terminal joints of the three middle fingers Diffuse swelling about fifth midphalangeal joint Thumb appears normal

B X ray shows marked narrowing, spurring and deformity of distal joints, especially of the three middle fingers Similar but less pronounced changes evident in mid-phalangeal joints, especially fourth and fifth fingers Metacarpophalangeal joints not involved In contrast to rheumatoid arthritis, bone adjacent to most affected joints shows increased density

motion is evident on physical examination Since the process is often more advanced on one side than the other, he can usually bend or rotate better to one side than to the other, although motion is more or less limited in all directions As a rule, forward bending is fairly well performed, in contrast to strain, fibrositis or myositis of the back which renders forward bending and straightening up difficult Dull ache or sharp pain in some other part of the body may be created by nerve root irritation From the cervical segment, it is referred to the head or shoulders, from the dorsal spine, to the chest or abdomen, from the lumbar spine, to the buttocks or legs It is usually aggravated by moving the back, and especially by coughing, sneezing, or other sudden motion When changes in the dorsal spine are pronounced, respiratory movement of the thorax is limited

Restriction of leg movements characterizes *hip* involvement If the process is unilateral, the patient will sit on one buttock If both hips are seriously diseased, he may not be able to sit at all In the advanced case, walking may be impossible but the patient will shuffle along with the aid of crutches.

Constitutional Disturbances. In contrast to rheumatoid arthritis, these are usually absent The patient feels well except for local symptoms The blood count and sedimentation rate are normal, although the latter is sometimes



FIG 35 24 Degenerative disease of knee Extensive spur formation without bone atrophy. Joint surfaces relatively smooth



FIG 35 25 Degenerative disease of spine Marginal lipping and increased bone density.

	RHEUMATOID	DEGENERATIVE
Age	Any. Eighty per cent between 20 and 60	40 and above
Onset	Variable	Insidious
Prodromes	Present	Absent
Fever	Often present	Absent
Tachycardia	Often present	Absent
General condition	Sick, undernourished	Often obese
Joint involvement	Generalized. Smaller joints	Localized Weight-bearing joints
Effusion	Common	Rare
Swelling	Soft tissue	Bony
Muscle atrophy	Present, often pronounced	Only in hip
Vasomotor changes	Present	Absent
Subcutaneous nodules	Present in 20 per cent	Absent
Sedimentation rate	Moderate to marked increase	Normal or slight increase
X-ray findings	Atrophic changes	Hypertrophic changes
Ankylosis	Frequent	Rare
Activity	May alleviate pain	Increases pain
Prognosis	Uncertain	Good

FIG 35 26 Table showing important differences between rheumatoid arthritis and degenerative joint disease (Prepared with assistance of Dr Walter Bauer)

slightly elevated The cutaneous changes characteristic of rheumatoid arthritis are lacking

X-ray Findings. One sees normal or increased bone density, narrowing of joint space, marginal proliferation (lipping, spurs, or osteophytes) and, rarely, a loose body

DISORDERS SOMETIMES CONFUSED WITH RHEUMATIC DISEASES

By creating pain, subjective stiffness, impairment of motion, or other symptom or sign suggesting trouble with a joint, a wide variety of disturbances are capable of being mistakenly diagnosed as arthritis In addition to those described in the following pages, chronic muscular or ligamentous strain of the back or an extremity, bursitis, tendonitis, sacroiliac strain, foot strain, ruptured intervertebral disc, primary or metastatic bone tumor, and spinal cord tumor or other neurologic disease should be mentioned

DISSEMINATED LUPUS ERYTHEMATOSUS

Disseminated lupus erythematosus, dermatomyositis, polyarteritis nodosa, and scleroderma are generalized systemic diseases which are often grouped together as the *collagen* or *connective tissue* diseases. Rheumatic fever and rheumatoid arthritis also belong in this category.

Disseminated lupus erythematosus is much more common in women, especially those between the ages of 20 and 40 Its clinical picture is extremely variable. Onset, as a rule, is marked by irregular fever, malaise, weight loss, and

arthralgia; consequently the disease in its early stages may be readily mistaken for rheumatic fever or rheumatoid arthritis. Although the course is marked by remissions and exacerbations, the disease is usually progressive and eventually fatal.

Because of the diffuseness of the pathologic process, symptoms and signs referable to almost any part of the body can develop. Changes in the skin and mucous membranes appear in most cases, sometimes at onset, often later (*see* Chap. 3). In a few cases they are lacking. Exposure to sunlight is likely to create an exacerbation, not only of the mucocutaneous lesions, but also of other manifestations.

Pains and aches in the muscles and joints, sometimes migratory, occur in roughly 95 per cent of patients. As a rule, these are out of proportion to the objective findings, but in perhaps one-third of cases clinical and x-ray changes indistinguishable from those of rheumatoid arthritis will develop. Other features are pleuritis, pericarditis, or peritonitis with or without effusion, lymphnodopathy especially of the cervical and axillary groups, enlargement of spleen or liver, pulmonary changes resembling those of primary, atypical pneumonia, some form of renal impairment, esophagitis, abdominal pain with or without diarrhea, gastro-intestinal hemorrhage, inconstant neurologic variants, local vasomotor changes, and retinal hemorrhages or exudates. Involvement of the myocardium may be indicated by tachycardia, gallop rhythm, indications of failure, or non-specific electrocardiographic variants. Warty vegetations on a heart valve are sometimes found at autopsy (*Libman-Sacks endocarditis*), but are rarely of sufficient size to be clinically diagnosed.

Although a few die of fulminating disease within a short time, most patients pursue a long course with gradual decline marked by remissions and exacerbations. Even during remissions low grade ill health may persist. As a rule, all of the manifestations indicated above do not occur at the same time, different ones appear during different exacerbations and may be transient. However, through the years the trend is for more and more systems to become permanently involved and for constitutional changes such as fever, anorexia, and weight-loss to increase. Death occurs from general decline, failure of a vital organ, or superimposed infection.

Important laboratory findings are anemia, leukopenia, thrombocytopenia, urinary variants reflecting renal damage, and elevated serum globulin with or without diminished albumin. False positive serologic tests for syphilis are likely. The diagnosis can often be confirmed by detection of so-called L.E. cells in specially prepared specimens of peripheral blood. Repeated tests may be necessary but failure to discover these cells even after multiple searches does not exclude the disease.

Dermatomyositis

A systemic disease of unknown cause, dermatomyositis is marked by inflammatory changes in the skin and subcutaneous tissues, skeletal muscles, and sometimes the viscera. Onset is usually insidious with vague complaints of low-grade fever, malaise, fatigability, or local or general muscular pain, stiffness, or weak-

ness Sometimes fever, prostration, painful muscles, and cutaneous eruption start abruptly.

The skin manifestations are diverse. Localized patchy or quite generalized erythema, scaling, and thickening, with later atrophy and pigmentation, are the most characteristic (*see* Chap. 3) The tendency is for these changes to predominate over large muscle groups. Early development of erythema and edema of the eyelids is likely; Raynaud's phenomena may occur.

The most striking symptoms are pain, stiffness, diminished strength, and tenderness of skeletal muscles At the start these may be local or general but subsequently show predilection for the trunk and the large muscle groups, especially the proximal muscles of one or more extremities Eventually, atrophy and contractures are likely; the former can be concealed by brawny edema of overlying tissues Diplopia, dysphagia, hoarseness, or dyspnea may reflect involvement of the pertinent muscles. *Arthralgia sometimes leads to a mistaken* diagnosis of arthritis but actual changes in the joints are not present, unless from contractures Among other possible features are non-specific lesions of the mucous membranes, retinal exudates, lymphnodopathy, splenic enlargement, *and mild neurologic variants Constitutional disturbances are not striking,* although sometimes loss of weight and persistent low-grade fever are encountered.

The disease progresses downward with exacerbations and remissions Duration is variable As a rule, death occurs from an intercurrent infection, especially pneumonia, from cardiac failure secondary to myocardial involvement, or respiratory failure resulting from impairment of the diaphragm and intercostal muscles.

Laboratory studies show mild anemia, normal leukocyte count with often an increase in eosinophils, monocytes, or both, and an elevated sedimentation rate. Increased urinary excretion of creatine is the rule Biopsy of muscle and overlying skin and subcutaneous tissue will show changes consistent with, but not pathognomonic of, dermatomyositis

Polyarteritis Nodosa

Also known as *periarteritis nodosa*, this disease is marked by irregularly and erratically distributed segmental inflammatory changes in the small and medium-sized arteries, and arterioles It has a predilection for males in mid-life. Although the cause is unknown, hypersensitivity is regarded as probably a contributing factor.

Vessels virtually anywhere in the body can be diseased. The clinical manifestations are extremely variable, since they reflect trouble in whatever organs happen to be affected by impaired blood supply. As in other diseases in this group, onset may be insidious or abrupt, with fever, malaise, and fatigability. Arthralgia, neuropathic pain, or myalgia during the early phase may lead to a mistaken diagnosis of rheumatic infection or rheumatoid arthritis

Depending upon the sites of the vascular lesions, one can find, as time goes on, one or more of the following features: hypertension; renal impairment; asthma, cough, dyspnea, pleural pain, or other indication of pleuropulmonary

inflammation; nausea, vomiting, diarrhea, or bouts of abdominal pain; evidence of peripheral neuropathy, such as paresthesia, sensory impairment, muscular pain or atrophy; pain, tenderness, atrophy, or impaired movement of one or more muscle groups; clinical or electrocardiographic evidence of myocarditis or pericarditis; headaches, cranial nerve palsies, seizures, papilledema or retinal hemorrhages and exudates, or cutaneous changes such as purpuric eruptions, erythema nodosum, urticaria, bullous lesions or areas of focal necrosis. Very rarely, one may by meticulous palpation detect small (2–3 mm), firm, subcutaneous nodules most likely in an extremity, usually along the course of an artery, these represent focal areas of vascular and perivascular inflammation. Since they are so infrequently found, they are, contrary to popular teaching, of little diagnostic significance. Failure to find them by no means excludes polyarteritis nodosa

Occasionally seen in an acute form, the disease as a rule runs a subacute or chronic course marked by exacerbations and remissions, not only of the systemic manifestations such as fever, loss of weight and strength, but also of the focal disturbances The trend is for symptoms and signs referable to different systems to appear during different exacerbations and to regress partially or totally during remissions Death occurs from gradual decline, or some form of visceral failure, most likely cardiac or renal

Laboratory studies show moderate anemia and, in contrast to disseminated lupus erythematosus, moderate to high elevation of polymorphonuclear leukocytes Increased eosinophile count is common, but it may be transient and detected only after repeated tests Sedimentation rate is rapid. If the kidneys are involved, albumin and other indications of renal disease will be found In a small number of cases, false positive serologic tests for syphilis are encountered If a lesion in the skin, subcutaneous tissue, or muscle is discovered, diagnosis may be established by biopsy but because the vascular lesions are so scattered, negative findings do not exclude the disease In a suspected case, even in the absence of nodules, random biopsies of muscle are sometimes indicated in an effort to establish diagnosis by finding the characteristic vascular lesions

SCLERODERMA

More common in women in mid-life, this disease is marked by changes in the skin, mild systemic disturbances, and, in the later stages, visceral lesions Onset is usually marked by slight fever, weakness, arthralgia, and perhaps local or diffuse edema Often a history of previously existing Raynaud's-like phenomena can be obtained Most striking are the cutaneous lesions which appear first on the face, neck, upper chest, or upper extremities; with time these parts may become almost completely involved and eventually virtually the whole body. Initially, the skin shows brawny edema but later becomes waxy, smooth, tight, and partially fixed to underlying structures. After months or years it is hard, atrophic and immobile, erythema, pigmentation, loss of pigmentation, anhidrosis, or depilation can develop. The underlying muscle is atrophic Trophic ulcers may appear in any diseased area. When the face is extensively affected, a mask-like

expression and even impaired mobility of the jaws will result. Involvement around joints can create difficulty of movement and contractures.

Dysphagia due to diffuse infiltration and impaired peristalsis of the esophagus, or stenosis of the lower esophagus, is likely. Occasionally, abdominal distress, cramps, or other digestive complaint reflects involvement of the stomach or small bowel. Delayed passage of barium, dilatation of the lumen, and other changes will be detectable by x-ray. Sometimes evidence of cardiac or pulmonary impairment is found As a rule, the disease slowly progresses for years, although occasionally it will spontaneously regress or become stationary In the severe, long-standing case one may find extensive contractures of the face or extremities, and malnutrition from fixation of the jaw or impaired function of the alimentary tract Death is most likely to result from an intercurrent infection superimposed on debility.

A focal form of scleroderma is sometimes seen; here, local areas of cutaneous change are found on the trunk, neck, and extremities, but the other manifestations just described are lacking.

Allergic Joint Disease

Within 7–10 days following injection of any of the animal sera, or administration of certain other therapeutic agents, especially penicillin, a sulfonamide, or a barbiturate, the patient may develop severe migratory polyarthritis, usually of the larger joints. Redness, swelling, increased surface temperature, and marked restriction of motion develop Other manifestations of allergic response are fever, severe urticaria, lymphnode enlargement, nausea and vomiting, and evidence of kidney irritation Regression within a week or 10 days is the rule, but occasionally duration is several weeks. There are no sequelae.

Blood Dyscrasia

Hemophilia. Bleeding into a joint occurs frequently in hemophilia, often but not necessarily, induced by trauma Pain, swelling, and impaired motion result. The blood is eventually absorbed but changes not unlike those of degenerative joint disease are a likely outcome following repeated episodes

Purpura. Pain with perhaps swelling and other signs of trouble may appear in one or more joints as a result of small intra-articular hemorrhages, or effusion. This is more likely to occur in the forms of purpura not associated with thrombocytopenia.

Sickle-Cell Anemia. Repeated episodes of muscle aches or arthralgia usually without objective evidence of joint change, especially when they occur in young Negroes, require exclusion of sickle-cell anemia Since the joint manifestations are likely to be associated with fever, and patients with severe disease may eventually develop myocardial impairment, the symptoms are sometimes wrongly attributed to acute rheumatic infection. Diagnosis can be established by demonstration of hemolytic anemia, and sickling of red cells in specially prepared wet smears of the blood.

Pulmonary Osteoarthropathy

The simplest form of this phenomenon (*clubbed fingers and toes*) and its causes are described in Chapter 6 In bronchopulmonary malignant or suppurative disease, a more extensive process is sometimes encountered. In addition to clubbed digits, one finds enlargement of the distal ends of the bones of the forearms, hands, wrists, and ankles; rarely, the larger bones of the extremities, the spine, and other skeletal bones become involved Joint pain, stiffness, soft tissue swelling, and impaired mobility create a picture readily confusable with arthritis If the underlying cause, especially when it is a mediastinal lesion producing pressure on the trachea or bronchus, develops rapidly, pulmonary osteoarthropathy may appear so acutely that it will be mistaken for acute rheumatic infection Occasionally x-ray changes will be found in the absence of clinical manifestations The x-ray picture is of *periosteal proliferation at the distal ends of the long bones, usually symmetrical and involving the upper and lower extremities to the same degree* The fingers and toes show soft tissue swelling of their tips, and tufting of terminal phalanges. The joint surfaces are normal If the underlying cause can be corrected, the clinical and x-ray manifestations will partially or totally disappear

Psychogenic Rheumatism

Backache, muscle aches or pain, stiffness or impaired motion of one or more joints is not infrequently encountered secondary to emotional stress Clinical, laboratory, and x-ray evidence of joint changes are not found Cases falling in this category are common in military personnel, especially during time of war when poor motivation is so often operative Lack of objective evidence of trouble serves to exclude the usual forms of arthritis To differentiate psychogenic rheumatism from fibrositis is extremely difficult and requires thorough evaluation of the case from the psychiatric as well as the clinical standpoint At times the diagnosis can be made by establishing the fact that the patient feels better in the morning after a night's rest, a circumstance which does not pertain in fibrositis, myositis, and most other musculo-skeletal disturbances

Occasionally, hysterical paralysis of long duration will be attended by sufficient periarticular fibrosis, and degenerative or atrophic changes of cartilage, to result in permanent joint damage.

Rachitis

This is a disease of infants and young children resulting most commonly from insufficiency of calcium, phosphorus, or vitamin D When encountered in adults the same type of deficiency is called *osteomalacia* Serum calcium is normal or low, phosphorus low, and alkaline phosphatase elevated The predominant skeletal change is improper calcification of the bones, particularly at their epiphyseal ends

In the young, suggestive manifestations of the active stage are *restlessness, irritability, head sweating, lack of muscular tone, malnutrition, or pain in the*

extremities, which may be confused with arthritis. The characteristic skeletal changes are softening of the bones of the skull (*craniotabes*), prominence of frontal and parietal bosses, enlargement of costochondral junctions which appear as rounded subcutaneous swellings beneath the skin to each side of the sternum (*rachitic rosary*), and enlargement of the epiphyseal ends of long bones. In the long-standing case the thorax becomes deformed by flaring of the lower ribs, depression above them which follows the line of diaphragmatic attachment (*Harrison's groove*), and perhaps pigeon breast The legs are bowed, knock-kneed, or otherwise deformed Kyphosis or other spinal curvature is not infrequent and may be so severe that the patient is dwarfed Other characteristic findings are pot-belly due to weakness of abdominal muscles, enlargement of liver or spleen, and anemia, probably resulting from general malnutrition rather than the disease itself. During the acute stage tetany may develop The bony deformities, especially squaring of the head due to prominence of the parietal and frontal bosses, pigeon breast, Harrison's groove, and misshapened legs, are often permanent.

In acute rickets, x-rays of long bones show widening of the epiphyseal lines, metaphyseal cupping, and perhaps diminished density of their shafts Later, permanent deformities are often demonstrable In osteomalacia, generalized skeletal decalcification which may cause bone pain is the one constant finding, bowing, spontaneous fractures, and dorsal kyphosis are sometimes encountered.

SCURVY

Effusion into one or more of the larger joints is not uncommon in infantile or adult scurvy Subperiosteal hemorrhage of a long bone in the region of a joint, especially when an associated effusion is present, may lead to an incorrect diagnosis of arthritis The same mistake may be made in a child following dislocation of an epiphysis.

HYPERPARATHYROIDISM

Parathyroid hypersecretion, usually due to adenoma of one gland, occasionally to primary hyperplasia of all four, or rarely to carcinoma, causes a disturbance of calcium and phosphorus metabolism With time, a high percentage of cases will develop skeletal changes (*osteitis fibrosa generalisata*), the symptoms of which may be confused with those of arthritis or neuritis. Since the bone changes are a late manifestation, it must be emphasized that hyperparathyroidism often exists without them. The disease is most common in the third or fourth decades. A similar picture can be created by secondary parathyroid hyperplasia in long-standing renal disease, such as chronic glomerulo- or pyelonephritis

Any of the following manifestations may appear first or develop as the disease progresses: lassitude, weakness, easy fatigability or weight loss, deep, boring pain in the long bones, spine or pelvis; spontaneous fractures; polyuria, nocturia or polydipsia due to increased calcium and phosphorus excretion, indications of renal impairment due to nephrocalcinosis; ureteral colic or signs of renal infection secondary to calculus, tumor of jaw, spreading of teeth, or develop-

ment of malocclusion or prognathism If the initial symptom is pain in a lower extremity, the case may be misdiagnosed and treated for foot strain or arthritis Polydipsia or polyuria may be erroneously attributed to diabetes insipidus.

Physical examination can be negative or show changes reflecting one or more of the variants indicated above. Band keratopathy may be found In the long-standing case, wedging of vertebrae secondary to demineralization can be manifested by body shortening, kyphosis or both *Clinical or laboratory indications* of renal impairment secondary to pyelonephritis, stone or nephrocalcinosis are usually found Indeed, discovery of nephrocalcinosis or a calcium stone always demands study of the patient to exclude hyperparathyroidism. In secondary hyperparathyroidism, signs of renal disease antedate those of parathyroid involvement.

The classic laboratory findings of primary hyperparathyroidism are elevated serum calcium, diminished phosphorus, increased urinary excretion of calcium and phosphorus, and, when bone involvement is moderate to pronounced, elevated serum alkaline phosphatase In secondary hyperparathyroidism, *phosphorus excretion is diminished so that serum phosphorus is elevated, serum* calcium is diminished, normal or elevated depending on the degree of acidosis and activity of the parathyroids

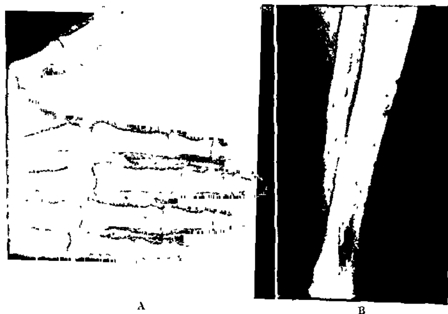


Fig 35 27 *Hyperparathyroidism*

A Generalized demineralization Marginal irregularity of phalangeal shafts and fraying of ungual tufts due to *subperiosteal resorption* (*Typical lace like pattern of marginal decalcification, evident on original film, lost in reproduction*) Terminal phalanx of index finger shortened. Middle phalanx of third finger shows an *irregular cyst-like area of diminished density*

B General demineralization of tibia and fibula with circumscribed areas of diminished density. (*Arteriosclerotic calcification of arteries also evident*)

When the bones are involved, diffuse demineralization of all or most of the skeleton will be evident by x-ray. Subperiosteal resorption creates a distinctive lace-like irregularity of the margins of the phalangeal and sometimes the metacarpal shafts, along with fraying of the terminal tufts, often enough to cause

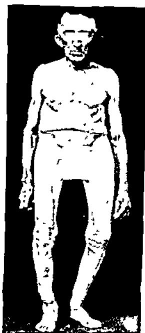
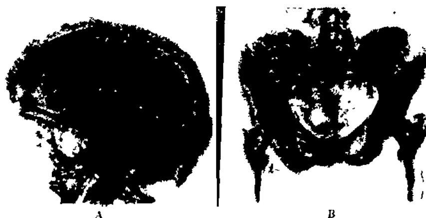


FIG 35 28 Advanced Paget's disease Striking features are enlargement of cranium with prominent forehead, body shortening, and bowing of thighs and legs (Courtesy Dr John B Stanbury)



A B

FIG 35 29 Paget's disease

A Lateral view of skull Mottling due to areas of increased density with interspersed areas of diminished density Thickening of inner table and mottling of diploic space apparent Thickening of outer table, evident on original film, lost in reproduction

B Pelvic bones Thickening of trabeculae and generalized increased density with scattered rounded areas of demineralization Involvement of left femur indicated by thickened cortex and slight bowing. Right femur essentially normal

actual shortening of the distal phalanges. A finding which may lead to relatively early diagnosis is partial to complete loss of laminae durae, observed on intraoral films of the teeth; when the jaws become well demineralized, the teeth—which do not lose calcium—stand out in bold relief. In the advanced case, one may find bone deformities, fractures, wedging of one or more vertebrae, or circumscribed areas of radiolucency representing cyst like cavities of fibrous tissue filled with fluid or tumor like masses composed of osteoblasts and osteoclasts.

Paget's Disease

Also known as *osteitis deformans*, this is a disease of unknown etiology occurring almost always beyond middle life and predominant in males. It is characterized by scattered areas of bone resorption and deposition affecting most commonly the skull, vertebrae, pelvis, and lower extremities. The entire skeleton is never involved. Trouble may be limited to a single area or be found in scattered areas in various parts of the skeleton Since the disease often exists without clinical manifestations, the diagnosis is frequently made incidentally by x-ray

Bone ache or pain in any part of the body, usually the back, pelvis, or lower extremities is the outstanding symptom Physical examination may be negative. Other cases show one or more of the following features characteristic bowing with resultant shortening of the lower and, sometimes, of the upper extremities, kyphosis of the spine which also contributes to loss of height, increased diameter of the skull, and prominence of frontal bosses which makes the size of the cranium out of proportion to that of the face Occasionally one will find unilateral, painless swelling beneath the gum, most likely in the posterior segment of the maxilla; this represents enlargement of one or more alveolar processes by the typical bony change Localized redness, swelling, and tenderness of skin and subcutaneous tissue resembling cellulitis are sometimes present overlying a diseased area of bone, as a result of increased vascularity in the region When the changes in the appearance of the patient are evident, the diagnosis can be made merely by looking at him Otherwise, it is usually established by x-ray The most important laboratory finding is pronounced elevation of the serum alkaline phosphatase Urinary calcium excretion is sometimes increased

By x-ray the involved area of bone will be enlarged and show thickening of its cortex Disease of the skull is indicated by expansion of the tables and rearrangement of the diploe into a coarse, mottled pattern Less often one will find areas of diminished density in the skull or long bones

The disease is rarely fatal but may lead to disability from mechanical difficulties, pain, or spontaneous fractures, the last-named are less common than in hyperparathyroidism Osteogenic sarcoma developing in a segment of diseased bone is an occasional complication. Since this tumor is predominant in the young, it should suggest underlying Paget's disease when found in an older person

36

THE NERVOUS SYSTEM

In this chapter we discuss abnormalities of sensation and muscular activity (*neurologic disorders*), *and of perception, thinking*, emotion, and behavior (psychiatric disorders) Neurologic and psychiatric diagnosis is based, to a large extent, on the same factors as medical diagnosis, namely a careful history, physical examination with special stress on neurologic signs, laboratory data, and x-ray studies In *addition, special procedures such as lumbar puncture*, pneumo- and electroencephalograms, arteriograms, and psychologic examination are often necessary

NEUROLOGIC HISTORY

The more common neuropsychiatric manifestations are change in personality or mental status, headache, pain, paresthesia, anesthesia or other sensory disturbance, impaired vision, vertigo, tinnitus, deafness, difficulty in talking or swallowing, gait disturbance, muscular weakness, paralysis, incoordination, twitchings or fasiculations, and convulsions.

Since some patients may be unable adequately to describe *Present Illness* or give a history, as in aphasia, amnesia, or coma, are unwilling to do so, as in depression, or are utterly unreliable, as in alcoholism or a psychosis, it is often necessary to go to a relative, friend, or other person for information Sometimes even when the history elicited from the patient appears reliable, confirmation of the facts is advisable However, a relative is sometimes no more dependable than the patient He may minimize, conceal, or exaggerate alcoholism, drug addiction, or peculiar behavior of the patient, or deny previous admissions to a mental hospital, encounters with the law, or asocial conduct

Particular points of importance in *Family History* are existence of manic-de-
ithies, Hunting-
Mental disease

In *Past History*, a story of trauma must be carefully evaluated. A history of any difficulty at birth or of past injury to the head is of special significance in any case of convulsions or impaired consciousness; *so is injury to the back, in* cases of backache, weakness or impaired sensation of an extremity, or other disorder which might be the result of spinal cord damage Yet one must not be misled by the stress which the family of a patient with mental disease lays

on some trivial accident of the remote past. In the case of a child with mental [illegible]

[illegible] stupor or convulsions may establish the etiology of later convulsions In Parkinsonian syndrome, careful questioning may bring out a story of a past illness consistent with encephalitis.

If a story of "nervous breakdown" is obtained, its exact nature should be determined; for by this term the patient or his family may be referring to a depressive psychosis or an anxiety neurosis, less often to schizophrenia, early general paresis, or multiple sclerosis.

The *Sexual History* may have to be inquired into in great detail Impotence in the male, though common in old age, general debility, and severe fatigue, may be due to psychologic inhibitions, neurosis or depression, or to tabes dorsalis, myelitis, or cord tumor In a woman, frigidity, although it may be created by a local pelvic disturbance, maldevelopment, poor technique, fear of conception, "incompatibility" or one of the cord diseases just mentioned, is much more likely to be due to neurosis or other psychologic variant Transient frigidity or impotence is most often due to fatigue, but is common in depression; a return of normal libido is a sign of improvement In many cases of anxiety neurosis, a history of coitus interruptus or other unsatisfactory form of contraception is obtained In fact, unsatisfactory sexual life and neurosis are often found together, the former is probably a symptom of the latter and not, as popularly supposed, its cause.

Illegally induced abortions must be specifically inquired for, these often constitute psychic trauma, even in the most stable women Large doses of abortifacients may cause serious illness ergot produces vertigo, paresthesias, and convulsions, apiol, the symptoms of neuropathy Illicit sexual relations or extramarital pregnancy may precipitate a serious emotional upset

What was said apropos *Habits* in Chapter 1, especially fatigue and the use and abuse of alcohol and drugs, is of particular importance in cases of suspected nervous system or mental disease

Changes in personality and mental status, stupor, coma, hallucinations, delusions, obsessions, and phobias are discussed later

NEUROLOGIC EXAMINATION

Much can be learned from a cursory inspection of the patient when he is first seen. Alertness of thought and response, personality characteristics, facial expression, bodily or facial asymmetry, deformity, paralysis, gait, posture, or speech can provide a clue to the diagnosis. Although examination of special parts is always necessary, often more is gained by watching the patient perform a series of routine activities, sometimes, only in this way will one detect for example,

hemiparesis, chorea, or Parkinsonian syndrome. Certain signs such as choked disc or a Babinski response are alone sufficient to indicate serious disease

Stupor and Coma

Stupor is a state in which response to outside stimuli is markedly reduced; coma, profound stupor in which there is no response whatever to outside stimuli —the patient cannot be roused. The cause is often difficult to determine, especially when, as often happens, the physician knows nothing of the antecedent history Common causes are.

Cerebral Vascular Accident. *Cerebral hemorrhage, infarction* and *embolism*, and subarachnoid hemorrhage are described in chapter 37.

Uremia. Onset is usually gradual Headache, nausea, and vomiting, drowsiness, and muscular twitchings and convulsive movements usually precede the stupor History of hypertension, kidney disease, oliguria, or polyuria may be available On examination, one finds the usual signs of renal insufficiency (*see* Chap. 32). Uriniferous breath is suggestive Diagnosis is confirmed by pronounced elevation of non-protein nitrogen in the blood

Hepatic Insufficiency. In the terminal stages of cirrhosis of the liver or acute *hepatic insufficiency, muscular twitchings, convulsions, and coma are often* encountered Flapping movements of the extremities, often seen in hepatic failure, are described in chapter 6 The symptoms and signs of liver disease are usually evident.

Diabetic Coma. This begins gradually and is often preceded by headache, apathy, and drowsiness. History of diabetes is usually obtainable, perhaps with a story of neglected diet, failure to take insulin, or recent infection Hyperpnea and acetone breath are suggestive The urine shows large amounts of sugar, with acetone and diacetic acid

Hypoglycemic Reaction. Diabetics being treated with insulin are always in danger of developing an *insulin reaction* It is most likely to occur soon after a dose of insulin, but may appear later, especially if the patient ate less, exercised more than usual, or used long-acting insulin. If the reaction is severe, unconsciousness may occur suddenly, or follow a short period of nervousness, irritability, *mental confusion, double vision, weakness, faintness, or generalized* seizures Unconsciousness does not occur in a mild reaction Common signs are dilated pupils, pallor, tremor of the hands, and cold, clammy extremities Signs of circulatory collapse may appear. The blood sugar is usually below 50 mg. per cent.

Recurrent attacks of a similar nature are encountered in the rare case of hyperinsulinism due to hypertrophy or tumor of the islands of Langerhans. Occasionally in hyperinsulinism, the patient will show episodes of peculiar behavior, each lasting for several hours. Repeated blood sugar tests may be required during an attack before the low level is detected.

Post-Convulsive Coma. This is suggested by the convulsions which precede it and the history, if obtainable, of previous similar attacks. If the patient is in

coma and a history is not obtainable, a bleeding tongue, scars from previous falls, or evidence of involuntary urination or defecation are suggestive.

Syncope. This is sudden and there is usually transient, partial, or complete loss of consciousness. It is usually due to a temporary change in the circulation from one of a variety of causes.

VASOVAGAL SYNCOPE (FAINTING ATTACK) This is the most common form. It rarely occurs when a person is lying down Fatigue, hunger, worry, emotional upset, standing for long periods of time, especially in a crowd or in a badly ventilated room, and weakness from recent illness or long confinement to bed are common precipitating factors. *Emotionally unstable women are more susceptible than others.*

Unconsciousness may come without warning or be preceded by a period during which one feels lightheaded, weak, hears sounds faintly, and has blurred vision Vasovagal syncope and syncope due to other disturbances of the circulation such as tachycardia, heart block, valvular disease, postural hypotension, and peripheral circulatory failure have been described in chapters 9 and 11. During a prolonged attack of unconsciousness, as in Adams-Stokes syndrome, convulsions may occur.

CAROTID SINUS IRRITABILITY. *Attacks of syncope may occur as a result of* stimulation of the sinus as by sudden turning of the head or pressure from a tight collar, less commonly from local disease such as cervical lymphnoditis or tumor. The attack may be due to slowing of the heart or fall in blood pressure, or occur without evidence of circulatory change Carotid sinus sensitivity is determined by pressing the fingers backward and medially toward the spine from a point in front of the sternomastoid muscle just below the angle of the jaw The normal response is slight slowing of the heart beat and slight depression of the arterial blood pressure In a person with a hypersensitive sinus, the respone *is faintness, syncope, or convulsive seizure usually but not necessarily* accompanied by slowing of the heart beat or pronounced lowering of blood pressure

Head Injury. Temporary unconsciousness lasting for a few minutes following a blow on the head is due to *concussion*, if it lasts longer than a few hours, it usually means *intracranial bleeding* or a *contusion* of the brain. Often the patient will regain consciousness after a few moments only to relapse later into drowsiness deepening into coma, from continued extra- or subdural bleeding Nausea and vomiting, slowing of the pulse, and gradual rise of blood pressure usually occur. In *fracture of the skull, it is the intracranial bleeding or, in cases* of basal fracture, perhaps direct injury to the brain which causes the trouble. Fractured skull is suggested by contusion or laceration of the head or, in basal fracture, leakage of frank blood or watery blood (dilution by spinal fluid) from an ear or the nose Neurologic signs depend on the extent and type of injury.

Any patient who, following a blow on the head, no matter how slight, becomes temporarily dazed, stuporous, or unconscious, must be carefully watched for several days because of the danger of slowly developing intracranial hemor-

rhage. A negative x-ray of the skull is of no particular significance: not all fractures are visible, and hemorrhage or brain injury can occur without fracture.

Brain Tumor. Stupor and coma are late manifestations A gradual onset of variable neurologic symptoms or signs is the rule In contrast to popular concept, headache is often a late symptom. Drowsiness and confusion precede the stupor Choked discs and focal signs are usually present by the time stupor has developed

Brain Abscess. The picture is essentially the same as that of brain tumor, but onset is more rapid There is almost always a history of a chronic suppurative process, particularly mastoiditis, sinusitis, lung abscess, or brŏnchiectasis

Acute Alcoholism. History of recent drinking and alcoholic breath are significant but in themselves are not diagnostic. Coma is rarely complete *The practice of attempting to revive patients in coma with some alcoholic drink and failure to consider the possibility of head injury superimposed on intoxication often lead to tragic mistakes.*

Drug Poisoning. The patient can usually be aroused to some extent Respirations are slow or irregular Signs of circulatory failure may develop Pinpoint pupils, scars of needlemarks on thighs or arms, or the discovery of a hypodermic syringe or laudanum bottle afford important clues to poisoning by an opium derivative In overdosage of barbituric acid derivatives, the patient can usually be roused, will mumble unintelligibly and relapse into deep stupor Supraorbital pressure or slapping the soles of the feet may help rouse him Nystagmus and absence of abdominal and tendon reflexes may be noted

Gas Poisoning. In poisoning by carbon monoxide or illuminating gas, the circumstances under which the patient is found make clear the cause of unconsciousness

Sunstroke. This occurs following extreme exposure to the sun and is accompanied by hyperpyrexia—106°–110°F or even higher Headache, dizziness, thirst, epigastric pain, and vomiting may precede the coma The skin is hot, dry, and flushed, muscular twitchings may be seen Sunstroke must not be confused with heat exhaustion, in which the skin is cold and clammy, temperature is not elevated, and coma not present

Malaria. Coma, usually preceded by a few hours of mental confusion or actual delirium and perhaps by convulsions, is common in falciparum malaria. These variants may be the first sign of trouble or appear after other indications of the disease are evident. In a malarial attack due to one of the other plasmodia, convulsions are occasionally encountered, but only in patients who are subject to seizures or who have had convulsions during other infectious diseases

Hysteria. There is usually a history of previous episodes of unconsciousness or of other hysterical manifestations, such as transient blindness, paralysis, or periods of amnesia A history of multiple surgical procedures, especially if the operative findings were meager or inconclusive, should make one suspicious. Onset of stupor usually occurs during an emotional upset, under dramatic circumstances, and almost invariably when other persons are present The patient is never hurt when he falls, appears to be in a trance, and will often respond

to suggestion, or suddenly pass from the stuporous state to one of weeping or thrashing about The hands make grasping motions, irregular, semipurposive movements of various parts of the body are observed Loss of pain sensation over all or part of one side of the body *may be demonstrable In cases of this* kind, one must always consider the possibility of psychomotor automatism, or post convulsive coma or confusion

The Cranium

Hydrocephalus and microcephalus *have already been mentioned Asymmetry* resulting *from congenital* maldevelopment or birth injury may indicate developmental or degenerative disease. Localized tenderness may mark the site of trauma, subdural hematoma, or, rarely, tumor or abscess; localized headache, if present, is a reliable lateralizing symptom A depression in the skull may be the cause of irritation of the underlying brain at that point A localized swelling

tion over an angioma or arteriovenous fistula may disclose a bruit, the patient himself may be aware of the sound.

The Spine

Examination of the neck and back and their common disorders have already been described (*see* Chaps 5 and 7) Weakness of back muscles occurs in poliomyelitis and other spinal cord lesions, progressive muscular dystrophy, and myasthenia gravis Tenderness of the spine may indicate the site of an injury, ruptured intervertebral disc, infection, or tumor

The Cranial Nerves

First Nerve. Sense of smell is tested by holding up to each naris, while the other is kept closed by digital pressure, various substances having strong, familiar odors, such as peppermint, camphor, and coffee The patient is asked to identify the various odors and to compare the acuity of smell on the two sides. Where greater accuracy is desired, the quantitative tests of Elsberg may be employed Since a certain number of otherwise normal persons lose sense of smell, only absence on one side is helpful in diagnosis *Anosmia*, when not due to a nasal disturbance such as acute coryza or atrophic rhinitis, is most likely to be found in midline meningioma in the region of the cribriform plate (*olfactory groove meningioma*), tabes dorsalis, and after fracture involving this region Otherwise it is of little importance

Second Nerve. Tests for visual acuity and fields of vision, and examination of the optic nerve have already been discussed (*See* Chap 4)

No neurologic investigation is complete without ophthalmoscopic examination and at least rough tests of acuity and fields of vision

Third, Fourth and Sixth Nerves. Ocular movements are tested by having the patient follow with his eye an object such as the point of a pencil as it is moved

from side to side and up and down. When the degree of paralysis is slight, it may be difficult to tell which nerve or muscle is affected; special tests by an ophthalmologist are required Diplopia appearing during the tests usually means some degree of paralysis Nuclear and supranuclear lesions may give complex disturbances Sixth nerve palsy indicated by inability to move the eye outward is of limited localizing value since it can occur with increased intracranial pressure from a number of causes In *peripheral* lesions of the third nerve, one finds ptosis of the lid, dilated fixed pupil, external squint, and a fairly immobile eyeball, the two last-named resulting from impairment of all motions except those controlled by the external rectus and superior oblique muscles In *nuclear* lesions of the third nerve, ptosis and pupillary change may or may not be present, and not necessarily all of the movements controlled by the nerve are affected Impairment of conjugate eye movements indicates a lesion somewhere along the cortico-pontine pathways, as in cerebral or basilar artery thrombosis. Inability to converge is brought out by having the patient tell time from a watch first when it is held at arms' length and then as it is gradually brought closer to his eyes, it occurs in encephalitis and vascular disease or tumor of the mid-brain With impaired convergence, the pupils do not contract normally when the eyes attempt to focus on a near object Limitation of upward conjugate movements points to a lesion involving the tegmen of the mid-brain, most likely infection or pineal gland tumor

Pupillary disorders and nystagmus are discussed in chapter 4; nystagmus, later in this chapter also

Fifth Nerve. Disturbances of the fifth nerve are more often manifested by sensory than motor variants Sensory impulses are conveyed from the face, most of the scalp, the conjunctiva, cornea, and mucosa of the mouth and nose Impaired sensation or loss of corneal reflex (provided there is no obvious seventh nerve paralysis to account for it) indicates trouble in the central or peripheral sensory pathways of this nerve

The *corneal reflex*—a wink response of both eyes elicited by touching the cornea with a wisp of cotton—has its sensory pathway in the fifth nerve, its motor pathway, in the seventh When the test is performed, the patient must be looking away from the stimulus, otherwise he will wink when he sees it In unilateral seventh nerve palsy, the wink response does not occur on the affected side of the face, it does occur on the unaffected side. In unilateral disturbance of the sensory pathway of the fifth nerve, because sensation is impaired, wink response does not occur on either side when the cornea on the affected side is tested; it occurs on both sides when the unaffected cornea is tested Absence of corneal reflex is a valuable early sign of fifth nerve disturbance since it may precede obvious sensory impairment of the face, or will be demonstrable in the stuporous patient who is incapable of recognizing or reporting loss of sensory acuity. In deep coma the reflex is bilaterally absent It is not impaired in trigeminal neuralgia.

It is in the distribution areas of the fifth nerve, especially its second and third branches, that the pain of trigeminal neuralgia (*tic douloureux*) is felt. Some

local disorder such as acute sinusitis and impacted or abscessed tooth is a likely cause of *pain in all or part of the same area.*

[illegible] the motor fibers will be indicated by impaired action of the [illegible] ness it will deviate toward the affected side *If deviation is not clearly* [illegible] weakness can be demonstrated by having the patient open his mouth while one palpates the condyles of the jaw with his forefingers *Normally the condyles* move forward when the mouth opens; if the muscles are weak, forward movement will not occur on the homolateral side.

Seventh Nerve. In *peripheral* or *nuclear* seventh nerve paralysis, homolateral flattening and sagging of the face is observed The forehead cannot be wrinkled, the palpebral fissure is widened, the eye remains open or closes incompletely, the naso-labial fold is shallow, the corner of the mouth droops, and in talking, whistling, or smiling the paralyzed side functions poorly or not at all. When paralysis is complete, diagnosis is obvious, when incomplete, one relies on consistent minor findings, such as slight drooping of the corner of the mouth However, flattening or asymmetry due to loss of teeth or other less significant cause must always be excluded Peripheral facial paralysis occurs most commonly as an independent disorder of unknown cause (*Bell's palsy*) Peripheral or nuclear palsy is also seen in tumor, particularly of the cerebellopontine angle, diphtheria, bulbar poliomyelitis, syphilis, middle ear disease, and caries of the petrous portion of the temporal bone In facial paralysis resulting from a lesion within the middle ear or from disease of the posterior fossa such as cerebellopontine angle tumor, taste sense is often lost *on the anterior two-thirds of the tongue* (Pure taste, which is not to be confused with flavor—a combination of *taste and smell—is tested for by putting on various parts of the tongue in turn* small dry samples of sweet, bitter, acid, and salty substances.)

When a lesion causing facial paralysis is supranuclear as in infarction, hemorrhage, or tumor involving the corticobulbar tract, the picture is the same, except *that in all but the most extreme cases the eye* can close to some extent and the forehead can be wrinkled (*central facial palsy*) If the lesion is in the cortical region, purposive facial movements are more impaired than the involuntary expressive movements such as smiling, in lesions near the thalamus, the reverse is true

In some disorders, such as progressive bulbar paralysis, bulbar poliomyelitis, diphtheric neuropathy, infectious polyneuropathy, and myasthenia gravis, weakness or paralysis of both sides of the face may occur. This bilateral involvement may be hard to recognize The most reliable sign is inability to close the eyes tightly, especially against resistance.

Chvostek's sign, encountered in tetany, is contraction of the muscles of the face on the homolateral side when the cheek is tapped along the course of the facial nerve (*see* Chap 4)

Eighth Nerve. Auditory acuity and the common causes of conduction and perception deafness are discussed in chapter 4

Ringing, whistling, or buzzing in the ear (*tinnitus*) is usually associated with degeneration of the auditory nerve due to advancing age, post-traumatic changes, or some toxic factor It may be induced by large doses of certain drugs, especially *quinine*, quinidine, streptomycin, and the salicylates. It also occurs in *acoustic tumor*, neurosyphilis, basal skull fracture, and other causes of nerve deafness.

A disturbance of the vestibular division of the eighth nerve or its tracts is one of the causes of vertigo. This term refers specifically to the sensation of persistent rotation—the patient within his environment or the environment around him. It must be distinguished from dizziness and lightheadedness which, strictly speaking, are feelings of wavering, unsteadiness or uncertainty of position without a rotative component Vertigo is most often encountered in acute alcoholism, motion sickness, labyrinthitis, Ménière's syndrome, cerebellopontine angle tumor, multiple sclerosis, vascular lesions of the brain stem, and toxic doses of the drugs just mentioned

During an attack of vertigo created by an *irritative* lesion of the labyrinth or its central connections, such as labyrinthitis or cerebellopontine angle tumor, nystagmus predominating toward the contralateral side of the head is present. *Past-pointing* is also a feature It can be brought out by the following test. The examiner holds an object at arm's length in front of the patient and asks him, with eyes open, to place his finger on it He is then instructed, with eyes closed, to raise the outstretched arm above his head and then bring it downward to touch the test object again, the procedure is repeated several times The normal person will always make contact or miss the mark by no more than an inch or so. The patient with labyrinthine or a related disturbance will bring his finger down well to one side of the test object, usually further and further away with each successive try As a rule this lateral deviation will be toward the side of the lesion

Added information concerning labyrinthine function is obtained by caloric and turning (*Barany*) tests which, since they require special training, are beyond the scope of this text Careful audiometric studies are also indicated, since impairment of the labyrinth or its connections is rarely found in the presence of normal hearing on the affected side, the auditory deficiency may involve only a few frequencies and be overlooked in any less precise test.

If some *destructive* process such as infectious labyrinthitis, tumor progression, or inner ear hemorrhage creates total loss of labyrinthine function, nystagmus, vertigo, and spontaneous past-pointing will disappear, here, one must rely on caloric, turning, and other special tests to determine the nature of the underlying lesion.

Ninth Nerve. Containing motor, sensory, and secretory fibers, this nerve is rarely involved alone, usually being affected along with the tenth and eleventh. Characteristic indications are loss of sensation in the posterior part of the pharynx and tongue, and loss of taste on the *posterior* third of the tongue. Pharyngeal reflex may be absent (*see* Chap. 4) This nerve is involved in diseases of the brain stem, such as infarction, tumor, and chronic meningitis Im-

pairment provides little help in localizing a lesion. *Glossopharyngeal neuralgia*, comparable in its clinical manifestations to trigeminal neuralgia, is precipitated by coughing or swallowing, starts on the side of the throat and radiates to the ear and sometimes to the side of the face and neck.

Tenth Nerve. The vagus nerve is a mixed nerve with many functions. Involvement may be indicated by palatal paralysis, regurgitation of liquids through the nose on swallowing, dysphagia, or disturbances of the larynx. Nasal speech is often the first and perhaps the only indication of palatal paralysis; when the latter can be observed, the uvula deviates toward the unaffected side. The gag reflex may be absent. The tenth nerve or its nucleus is affected in diphtheric and infectious neuropathy, and in diseases of the medulla such as tumor, multiple sclerosis, bulbar poliomyelitis, and progressive bulbar paralysis. Tumor or operation in the neck may affect a recurrent laryngeal nerve, causing hoarseness or aphonia; disturbances due to intrathoracic tumor and aneurysm have previously been described. In myasthenia gravis, weakness of the pertinent muscles may create difficulty in swallowing or speaking comparable to that caused by a tenth nerve derangement.

Eleventh Nerve. The sternocleidomastoid muscle and the upper part of the trapezius are controlled by this nerve. Trapezius weakness is indicated by diminution of power to elevate the shoulder, sternocleidomastoid weakness, by impaired contraction of the muscle and diminished power when the patient attempts to rotate the chin toward the *opposite* shoulder. Since the sternocleidomastoids are bilaterally innervated but the trapezius is innervated *only from* the opposite cerebral hemisphere, a central lesion will impair the trapezius while the sternocleidomastoid will be transiently involved, if at all. On the other hand, in lower motor neuron lesions, both muscles will be uni- or bilaterally impaired. The eleventh nerve may be affected in diseases of the medulla listed above.

Twelfth Nerve. Normally the tongue protrudes in the midline although slight deviation to one side is not uncommon. Unilateral paralysis of the twelfth nerve is indicated by deviation of the tongue toward that side, when it is protruded. In pseudo bulbar palsy there is bilateral involvement of the pathways from the cerebral cortex to the twelfth nerve nuclei; here, on voluntary effort, the tongue may be protruded slowly and slightly, or not at all. On the other hand, it can sometimes be moved fully by reflex action from lower levels; for example, if one stimulates the upper lip, the tongue will protrude in response to the irritant. Atrophy and fasciculation occur in nuclear lesions such as progressive muscular atrophy. Slowness and awkwardness of tongue movement are found with cerebellar lesions, and dysarthria secondary to cerebral disorders. Tremor of the tongue occurring after several minutes' protrusion merely means fatigue. Disorders which involve the ninth and tenth nerves may affect the eleventh and twelfth nerves.

Motor System

Gait. Peculiarity of gait may be obvious or brought out only by such tests as running, walking on the toes or heels or along a straight line.

A *spastic* gait almost always means involvement of the corticospinal tracts and is found in subacute degeneration of the spinal cord, multiple sclerosis, spinal cord tumor, myelitis, and cerebral hemiplegia or diplegia In Parkinsonian syndrome, the patient shuffles along stiffly, without the natural swinging of one or both arms and shows other variants described in chapter 3. An unsteady, staggering or reeling gait may be due to general weakness, drug poisoning, alcoholism, neuropathy, or prolonged disuse of the legs. In *cerebellar incoordination* due to disease of the cerebellum or its tracts, such as acoustic tumor, cerebellar tumor, or Friedreich's ataxia, the gait is affected because of trouble in the motor coordinating apparatus, the patient walks equally poorly with eyes open or closed In *sensory ataxia*, as in tabes dorsalis, the trouble is in the posterior columns, the patient walks well if he watches the ground but, since he uses his eyes as a substitute for position sense, walks poorly in the dark In certain diseases such as multiple sclerosis and Friedreich's ataxia, both sets of tracts may be involved

Diseases in which involuntary movements are prominent such as Sydenham's chorea and Huntington's chorea show lurching, twisting and other oddities of gait *In paralysis of the peroneal muscles, as in pressure palsy of the peroneal* nerve, anterior poliomyelitis, or polyneuropathy, the foot cannot be dorsiflexed (*foot drop*), so that the leg must be lifted high in walking (*steppage gait*). In hysteria the gait does not correspond to any known pattern of neurologic disintegration, the patient may drag one or both feet, walk in a dramatic fashion, limp, stumble frequently, demand support, fall after taking a few steps, or even refuse to try walking Hysterical gaits are likely to remain remarkably constant.

Posture. In Parkinsonian syndrome or other disease of the basal ganglia, one observes a typical posture with head and trunk bent forward, to one side, or, rarely, backward, knees slightly flexed, and arms held rigidly at the sides even during walking Marked lordosis, with the abdomen protruding and the head back, is seen in progressive muscular dystrophy and in anterior poliomyelitis involving the back muscles Depressed patients may sit with head bowed and shoulders stooped in an attitude of dejection The catatonic patient will sit or stand for hours in a rigid, bizarre pose In the *decorticate* state due to a lesion *such as infarct or hemorrhage of the internal capsule or higher,* the patient lies in bed with his arms rigidly flexed and his legs extended, in the *decerebrate* state due to a lesion isolating the midbrain from the cerebral hemispheres, the arms are extended and internally rotated, and the legs extended Other peculiarities of posture have been described in chapter 3

Articulation. Speech difficulties are often noticed during the course of ordinary conversation, sometimes only during repetition of test phrases A monotonous, sing song, scanning, or jerky speech may occur in advanced multiple sclerosis and in cerebellar disease In general paresis, there is slurring; "methodist episcopal" is pronounced "messodis epistobal," for example. Slow speech is characteristic of *hypothyroidism*, Parkinsonism, drug poisoning, and states of impaired consciousness. Parkinsonian speech is monotonic, perhaps stuttered,

and, in the late stages, usually faint. Nasal speech, well brought out by the phrase "plum pudding," when not due to a local lesion of the nose or throat, means palatal paralysis.

Aphasia is described below.

Dominance. Whether the patient is right-handed (*right dominant*) or left-handed (*left dominant*) must be taken into consideration in comparing the strength of the upper extremities. Aphasia, in a right-dominant person, almost always means that the lesion is on the left side of the brain; in a left-dominant person, on the right side of the brain. Persons who stammer, mirror-write, or have difficulty in learning to read may have mixed dominance, as evidenced by ambilaterality.

Skilled Activity. Various causes account for difficulty in performing skilled acts—spontaneous activity such as dressing or undressing, or test activity such as threading a needle or striking a match Where muscle tonus is increased, as in Parkinson's disease, skilled acts are performed slowly; in cerebellar disease, jerkily. In weakness or paralysis of an extremity, they are hampered by restricted mobility or lack of voluntary control. In schizophrenia, depression, or malingering, the patient may not cooperate. *Apraxia* is inability to perform a skilled act in compliance with a request, due, not to paralysis, weakness or ataxia, but to impaired function at the level of cerebro-cortical coordination.

Tremors. Tremors of the mouth, tongue, and hands may be due to various causes, most commonly: cold, old age, extreme fatigue or debility, delirium, excessive use of alcohol or drugs, thyrotoxicosis, Parkinsonian syndrome, multiple sclerosis, cerebellar disease, general paresis, hysteria, and agitated or neurotic states In cerebellar disease and multiple sclerosis, tremor is accentuated in purposeful movements (*intention tremor*).

Fasiculations. These are fine or coarse twitchings of small bundles of muscles in the face, tongue, trunk, or extremities. They usually indicate slow degeneration of the anterior horn cells and are noted especially in progressive muscular atrophy and syringomyelia or syringobulbia. Sometimes they occur transiently in normal persons.

Choreic Movements and Athetosis. (*See* Chaps. 3 and 6)

Tics. These are fairly complex movements always repeated in the same definite patterns Common ones include spells of rapid blinking, twitching or contraction of one side of the mouth, jerking of the head to one side, and various respiratory deviants such as grunting, hacking, or sniffing Usually they are habit phenomena and most often develop in the tense or chronically fatigued. Rarely they occur from peripheral nerve irritation or as a late sequela of encephalitis. Often the cause cannot be determined

Seizures. Also called *epilepsy* and the *convulsive disorders*, seizures are manifestations of episodic discharges of groups of nerve cells which interfere with normal brain activity. Such attacks are to be regarded as a symptom precipitated by various stimuli and traceable to any number of causes. They vary in frequency, pattern, and intensity. A seizure may be marked by complete loss of consciousness with or without tonic or clonic convulsions of a group of mus-

cles or all the muscles, an unusual behavior pattern, development of some particular symptom such as abdominal pain, or a transient dream state. They may have a hereditary background, or one of congenital malformation or birth injury of the brain. They can also appear in brain tumor, injury, vascular accident, edema or infection, hypertensive or lead encephalopathy, long-standing alcoholism during a withdrawal period, meningitis, malaria, uremia, hypoglycemia, eclampsia, serious heart block, carotid sinus irritability, other less common disorders, and following administration of a convulsive drug such as strychnine or Metrazol. However, in approximately 50 per cent of cases of recurrent seizures, no cause can be discovered clinically or pathologically (*idiopathic epilepsy*).

In general, epilepsy beginning in childhood or early adult life is more likely idiopathic, whereas that which starts later is more likely due to some demonstrable cause. Convulsions are not uncommon during the acute infectious diseases of infancy and early childhood. Although they do not necessarily predicate seizures in later life, it is believed that the child who experienced them under such circumstances is more likely to have them subsequently than one who did not.

Since events at the start are often helpful in localizing the underlying lesion, the onset of an episode is, from the diagnostic standpoint, its most important phase For example, twitching of a group of muscles with perhaps spread to adjacent groups (*Jacksonian seizure*) suggests a lesion of the motor cortex, a localized sensory variant such as paresthesia or anesthesia, a parietal cortical lesion, visual phenomena, an occipital lobe lesion These manifestations appear contralaterally A sense of a peculiar odor or a bizarre psychologic state in which things appear unreal or strangely familiar bespeaks trouble in the temporal lobe Careful questioning of the patient and of by-standers who observed the onset of an attack is essential to elicit any of these clues.

Most dramatic is the *grand mal* convulsion In many cases the patient will first experience a strange feeling (*aura*) such as a vague sensation in the epigastrium, a paresthesia or other sensation in his thumb, toe or tongue, a visual, auditory or olfactory phenomenon, or twitchings of an extremity or one side of his face. Almost immediately he loses consciousness and falls to the floor in a state marked by generalized muscular rigidity (*tonic phase*) with apnea, cyanosis, and salivation After a moment the rigidity relaxes and is followed by intermittent, violent, jerking movements of the entire body (*clonic phase*) Respirations become stertorous Frothing at the mouth is the rule, involuntary urination or bowel movement may occur Biting of the tongue is likely After 2–4 minutes motor activity subsides but the patient remains in coma, stupor, or deep sleep which may last for several hours Following return to consciousness, confusion, headache, a sense of fatigue, and sometimes nausea and vomiting will continue for hours longer. Sometimes there is no aura Without warning abrupt loss of consciousness occurs, followed by the chain of events just described

At the other extreme is the *petit mal* attack characterized by abrupt, momentary lapse of consciousness without a fall or other phenomenon. Afterward the

patient may be totally unaware of the lapse or only know that he has missed a few words of a conversation. The by-stander will note *momentary staring of the* eyes, fluttering of lids, smacking of lips, blanching of face, or observe that the patient was briefly out of touch with his environment. Although petit mal *seizures tend to appear in early* childhood and disappear toward puberty, a certain number of patients will continue through adult life to have petit mal, grand mal, psychomotor automatism attacks, or any combination of these.

Perhaps least well recognized is the *psychomotor automatism*, most often due to a disturbance in the temporal lobe. Here the patient will experience specific illusions, hallucinations, thoughts or memories, repeatedly speak short phrases, perform simple acts, or wander about in a daze talking irrationally. He may even struggle with or strike at those who attempt to quiet him. *In each attack* his pattern of thought or activity is the same Because the start and cessation of the episode are gradual, subsequently the patient usually knows that he had an abnormal experience but is unaware of his actions. The striking feature of psychomotor automatism is the similarity of the pattern in each seizure. This type is often wrongly attributed to an outburst of temper or hysteria and less often is mistaken for the confused state associated with hypoglycemia in adenoma or hypertrophy of the islands of Langerhans An abnormal electroencephalogram will be found in psychomotor automatism, not in hysteria.

In addition to the three just described, there are a number of other forms of epileptic seizures which do not have such clear-cut manifestations Whatever the pattern, complete investigation is indicated in an effort to discover a remediable cause Depending on the nature and extent of the lesion, physical examination may show no variants, slight but significant abnormalities such as slight underdevelopment of one side of the body or mild accentuation of reflexes on one side, or striking neurologic changes which enable one promptly to focalize the trouble In a person subject to seizures, an attack may be induced by high fever, several minutes of overbreathing, flickering lights (*photogenic epilepsy*), anoxia, alkalosis, certain other changes in body chemistry, or administration of a convulsant drug

The course of the disorder is subject to wide variation. If there is an underlying correctable disease such as meningioma or hypoglycemia, seizures may be completely stopped by proper therapy When due to a progressive process such as malignant brain tumor or terminal uremia, attacks may be expected to occur *with increasing frequency* In idiopathic epilepsy one finds, in different patients, wide differences in the number and types of seizures; frequency also varies with the type. Petit mal as a rule will occur 5–10 times daily but the number may be as high as several hundred Grand mal and psychomotor episodes vary as a rule from several a week to only one in a year or more As already indicated, petit mal may cease during late puberty and the patient will have no further trouble, or it may be replaced by one of the other types, or a combination of them, including petit mal If petit mal seizures are occurring daily, one grand mal episode may be followed by pronounced reduction of the former for an appreciable time At the start grand mal is most likely to occur during the night.

Any form is subject to remissions and exacerbations, the latter tending to appear during periods of fatigue, emotional stress, alcoholic excess, and in women during the premenstrual week. A serious hazard is that the patient may at any time develop a series of consecutive attacks at short intervals with no intervening return to consciousness (*status epilepticus*), here death presumably from anoxia or massive adrenal hemorrhage is a possibility. Contrary to popular opinion, mental deterioration is likely only when there is underlying brain disease or if brain damage is created by anoxia or some other factor during an episode of status epilepticus. In most cases pronounced suppression of attacks can be effected by administration of appropriate drugs Once the patient is on an established program of therapy, too rapid reduction or withdrawal of medication may be followed by development of status epilepticus.

The sudden appearance of convulsive seizures in an adult, especially beyond mid-life, should make one suspicious of brain tumor

Associated Movements. Diminution or absence of associated movements, such as swinging arms when walking, bringing into play the muscles of facial expression when talking, and rotating the head toward the shoulder when turning around, is not infrequently one of the earliest signs of neurologic disease. For example, in slight hemiplegia, failure to swing an arm in walking may be the only obvious sign; in the Parkinsonian syndrome, a mask-like expression

Muscle Hypotonia. Most likely to be found in disease of the cerebellum or its tracts and on the same side as the lesion, this is indicated by hypermobility of the joints on passive movements, most apparent in the more proximal joints of the extremities Another sign of hypotonia is the *pendular* patella or triceps reflex (*see below*)

Muscle Weakness. A feeling of muscle weakness, heaviness, or fatigue often precedes demonstrable change General muscle weakness may occur in any acute or chronic disease, and from disuse

The various muscle groups are tested by having the patient attempt to move a part first without the influence of gravity, then against gravity, and finally against resistance applied by the examiner Routinely one tests movements of eyelids, facial muscles, head, back, extremities, and ability to grip

Weakness of a particular group of muscles is most likely due to disuse from any cause or to disturbance somewhere in the nerve pathways which control it. In myasthenia gravis, one or more are weak but the striking characteristic is their easy fatigability on use Most frequently affected are the ocular movements, chewing, and swallowing Involvement is often asymmetrical The other myopathies are also featured by predominating weakness of particular groups

Paralysis. In *partial* paralysis, the muscle is weak, in *complete* paralysis, it does not contract at all Paralysis of a limb in a stuporous or comatose patient can be detected by the following procedures·

1 Lift, then drop each of the four extremities in turn. A paralyzed extremity will fall more limply than a non-paralyzed one

2. Pinch or prick each limb in turn An unaffected limb will almost always move; a paralyzed one, not.

3. Place both lower or upper extremities in awkward or unnatural positions. An unaffected limb will move to a more natural position; a *paralyzed limb will not move.*

FLACCID PARALYSIS Occurring chiefly in disorders of a peripheral nerve or the anterior horn cells, this is seen in neuropathy, nerve injury, poliomyelitis, and progressive muscular atrophy. It is accompanied by diminished or absent tendon reflexes and later, muscle atrophy.

SPASTIC PARALYSIS. This occurs with lesions of any part of the corticospinal tract. It is accompanied by increased tendon reflexes, a positive Hoffman sign *if the arm is affected,* diminished or absent *superficial abdominal reflexes,* and, if the leg is affected, a Babinski sign. It is seen in cerebral vascular accident, brain tumor or abscess, multiple sclerosis, subacute combined degeneration of the spinal cord, cord tumor, and other less common disturbances.

Atrophy. The muscle is shrunken and shows loss of normal contour, firmness and resiliency. Atrophy is most pronounced in disease of the anterior horn cells such as anterior poliomyelitis, neuropathy, and following nerve injury. It also occurs in a variety of other disorders, including other forms of paralysis, arthritis, and disuse from any cause.

Hypertrophy. Vigorous use causes a muscle to grow larger, firmer, and stronger In progressive muscular dystrophy, certain muscles are atrophied; others, especially those of the calves, are either hypertrophied or appear to be, but in either instance, they eventually become weak.

Coordination. Impairment of coordination may be indicated by failure to perform with dexterity such routine acts as buttoning the coat, striking a match, writing, or using a knife and fork When not due to weakness or stiffness, it is most likely the result of diminished position sense (*sensory ataxia—see below*), or disease of the cerebellum or its tracts (*cerebellar ataxia*) on the ipsilateral side. Sensory ataxia occurs only in the absence of visual help. If the patient is permitted to watch his movements he can guide them visually and thus compensate for his impaired sense of position In cerebellar ataxia there is inability accurately to gauge or control the degree of movement required to perform a simple act (*dysmetria*), failure of antagonistic muscles to work in unison (*dyssynergia*), or a combination of the two Since the fault is lack of muscular control, incoordination will occur even when the patient attempts to make use of visual guidance In general, if the trouble predominates on one side of the body, the cerebellar lesion is on the same side

Cerebellar ataxia may be indicated by any of the following phenomena.

NYSTAGMUS Oscillatory horizontal movements of the eyeballs occur when the patient looks to one side, especially if he attempts to keep his gaze fixed on an object in the lateral range. As a rule, there is a quick jerk toward the point of fixation but the eyes repeatedly tend to drift back toward the midline and then again jerk laterally. This variant is more pronounced when gaze is directed toward the side of the lesion. In cerebellopontine angle tumor, nystagmus occurs only when the eighth nerve or the vestibular nuclei are encroached upon. (A medullary lesion involving the vestibular nuclei will also cause nystagmus.)

Vertical oscillation, usually greatest when the patient looks upward, is sometimes encountered in cerebellar disease, usually as a reflection of a lesion in the *vermis* It is more apt to be caused by trouble in the medulla such as multiple sclerosis, a vascular lesion, or tumor

Ocular Dysmetria The examiner holds his forefingers about a foot apart at arm's length in front of the patient at eye level The latter is then instructed to shift his gaze back and forth from one finger to the other In the normal person, the eyes will be observed to focus directly on each finger in turn as the gaze is shifted In cerebellar disease, they will "overshoot" the mark on the side of the lesion—that is, they will move further laterally than is required for a direct view; in attempting to correct the error, they will then momentarily oscillate before coming to a focus on the proper finger.

Dysarthria Defective tongue and lip movements, especially when consonants are being formed, cause slurring of words and syllables (*slurred speech*). Articulation is jerky and explosive, the voice often louder than normal Sometimes words are pronounced syllable by syllable (*scanning speech*).

Intention Tremor When the patient attempts a voluntary movement uncoordinated contraction and relaxation of opposing muscles create a tremor which becomes more pronounced as the movement proceeds and greater effort is made to control it.

Decomposition of Movement In an action requiring use of an entire limb, movements involving its various joints occur in sequence, not synchronously. For example, in reaching for an object the upper arm will move first, then the forearm, the hand, and finally the fingers

Dysmetria of Extremities If the patient is asked to point to or touch a near-by object, lack of control and ability to gauge movement will cause his finger to deviate laterally or vertically from the target This will also happen in sensory ataxia but, as indicated above, only when the task is tried without visual help In cerebellar ataxia, the finger will deviate usually to the side of the lesion even when the patient is permitted to watch his movements Similar tests can be used with the toes and heels

Faulty Finger-to-Nose Test. The patient is instructed to lift his arms to shoulder level and touch the tip of his nose with one, then with the other forefinger The procedure should be repeated several times, both slowly and rapidly, first with the eyes open, then closed Normally, one is able to put the finger accurately on the end of the nose, in ataxia, the mark will be missed by an appreciable distance. As in other coordination tests, distinction between cerebellar and sensory ataxia is made by determining the influence of visual guidance The finger-to-nose procedure may also bring out intention tremor and, in more severe cerebellar disease, decomposition of movement

Adiadochokinesis Awkward execution of rapidly alternating movements is brought out by having the patient alternately pronate and supinate both forearms as quickly as possible by striking the palms and backs of his hands on the anterior aspects of his homolateral thighs. In the lower extremities, difficulty can be brought out by having him tap the floor with his feet. To avoid over-

sight of mild unilateral impairment, corresponding extremities should also be tested separately: if they are tested together, slight slowing due to awkwardness on one side, may set a slower pace for the other so that a slight difference between the two will be overlooked. If the unimpaired extremity is tested alone, its pace will be faster than in bilateral performance

REBOUND PHENOMENON The patient, with arm outstretched, is asked to flex it so as to touch his chin with his hand while the examiner applies resistance by pulling the wrist in the opposite direction In the normal person, if one suddenly lets go, the motion of the arm will promptly cease; in cerebellar disease the subject is unable to check his flexion movement which will continue even to the point of his striking himself in the face. To prevent possible injury, it is advisable for the examiner to place his other arm between the patient's chin and the arm which is being tested.

FAULTY HEEL-TO-SHIN TEST While lying on the table the subject is instructed to place one heel on his opposite knee and run it rapidly and precisely down the front of his shin, then to try with the other heel and shin Repetition at varying speeds is indicated The normal person is able to perform the maneuver smoothly and accurately, the patient with ataxia, not Here too, sensory and cerebellar ataxia must be differentiated by noting the effect of visual control.

In the patient with severe cerebellar incoordination, movements of the trunk and extremities are not synchronized, the gait is reeling or staggering, the patient flings his legs, lurches, and often falls irrespective of whether his eyes are open or closed.

LANGUAGE AND RELATED FUNCTIONS

Weakening or loss of the faculty of language in any of its forms such as speaking, writing, reading, or understanding, due to impairment of association areas of the cerebral cortex is known as *aphasia*. As a rule, it occurs only when there is trouble in the dominant hemisphere (left hemisphere in a right handed person and *vice versa*) Because of the many interconnections between the association areas, a pure form of aphasia is rare; for example, a disturbance of speaking is rarely encountered without some evidence of trouble in one or more of the other spheres Except when the responsible brain lesion is exceptionally small, overlapping creates multiple difficulties in the fields of reception, expression, and thought integration

Before deciding that a patient is aphasic, it is highly important to exclude, (1) a motor disturbance affecting articulation as in bulbar palsy, or writing as in cerebellar ataxia; (2) sensory loss, such as blindness, deafness, or diminished touch and position sense as in peripheral nerve damage; (3) general mental impairment as in mental deficiency, stupor, dementia, or psychosis.

In evaluating a patient with aphasia, one must take into consideration his education, intellectual level, and possession of any specialized capacity as for a foreign language, mathematics, or music As a rule, areas in which one is less proficient are more vulnerable than others. For example, the person who speaks

a foreign language may have this more affected that his native tongue. On the other hand, if one is highly trained in a specific field, slight aphasia might well be overlooked by the ordinary observer but detected by someone skilled in the same specialty. Furthermore, even in the presence of definite impairment, a patient may, under a special circumstance such as emotional stress, momentarily appear to function normally; he may, for example, briefly speak correctly—perhaps an appropriate word or one of two sentences

Aphasia must always be looked for when trouble is suspected in the dominant hemisphere In the pertinent case it is essential to examine performance in talking, writing, understanding spoken language, identifying objects by the various senses, and perhaps accomplishing simple acts This is necessary because if there is doubt about existence of aphasia in one sphere, it may be apparent in another Furthermore, impairment in a particular field suggests trouble in a specific area of the brain—for example, a speech disorder, a lesion in Broca's area of the frontal cortex. However, the interrelations and overlapping mentioned above make it impossible always to pinpoint a lesion on the basis of the deficiencies detected.

The methods of testing performance, the indications of impairment, and the areas of the brain in which trouble is to be suspected are discussed below Obviously the more complex the test required to bring out difficulty, the less serious the aphasia, but this does not necessarily indicate that the underlying disease is less severe. Since poor performance may be emotionally disturbing to the patient and hamper his further efforts, it is best to proceed from the simple to the more complex tests Furthermore because of his easy fatigability he should be examined in repeated, short sittings, rather than submitted to a single, long ordeal. If mild impairment is suspected, the physician can use his ingenuity in devising complex tests which might bring out trouble not evident during routine procedures.

Even though a person with aphasia may not be able to speak, it must be remembered that he may understand what is said For this reason, *the physician must be on guard to avoid making, in the patient's presence, any discouraging statement about his condition; a carelessly dropped remark could well be a source of emotional trauma.*

Speaking. Converse with the patient, pose simple questions, or point to and ask the names of familiar objects In the most severe case he may indicate by facial expression or gestures that he understands the questions and knows the answers but cannot reply in words; or difficulty will be apparent in conversation as jumbled, meaningless, fragmentary jargon, use of incorrect words, omission of unimportant words such as articles (*telegraphic speech*), or hesitancy while he searches for the right words In the mildest case one may find only omission of a word or two or use of a wrong word as the patient is asked to repeat a series of consecutive sentences Impairment of speaking suggests a lesion in Broca's area of the frontal cortex

Understanding of Spoken Language. Start with simple instructions such as to open the mouth, close the eyes, raise the left hand, or point to a near-by

object. Serious trouble will be indicated by failure to perform at all, doing the wrong thing, or repetition of the first act as others are subsequently requested (*perseveration*). Less obvious difficulty may be discovered by requesting that two of these simple acts be performed at once, or several in sequence. In the mildest case the only indication will be inability to follow a fairly long conversation. Impaired understanding of spoken language suggests a lesion of the temporal lobe in the area around Heschl's gyrus.

Writing. Ask the patient to copy printed material first when it is before him and then to reproduce it while it is out of sight; to transpose some printed words to script; to write words which one slowly dictates, to write spontaneously. Allowance must be made for poor penmanship due to weakness or paralysis of the dominant hand, or unaccustomed use of the other. If one of these is thought to be a factor, a large crayon should be tried. Impairment will vary from inability to write at all or to proceed beyond the first letter, to minor slips in spelling or grammar The principal area concerned with writing is in the frontal cortex just above Broca's area. However, writing is regarded as the most complex of language functions so that deficiency in the field is present in many forms of aphasia and consequently has little localizing value

Reading. Place before the patient a series of brief, printed instructions to perform simple acts such as to touch his nose, point to the clock and pick up the pencil. The severely handicapped may pay no attention or indicate by gestures his awareness that something is expected of him but that he cannot understand what he sees If printed instructions are properly executed, trouble may perhaps be detected by the use of written words If these are properly interpreted, more complex directions in printing or writing should be given, such as to put the red book on the table but not to put the pencil in the tray Impairment might then be indicated by overlooking the "not"; the patient will move both the book and the pencil In the equivocal case one may ask the patient to read a paragraph and give its meaning Predominant trouble with written language should lead one to suspect a lesion in the parietal lobe near the tip of the angular gyrus.

Visual Identification. Loss of the perceptive faculty which enables a person to recognize the nature of objects by one of his senses is known as *agnosia—visual, auditory* or *tactile*, as the case may be It suggests a lesion in the parietal, temporal or, less likely, the occipital lobe.

To test for visual agnosia, point to and ask the patient to name various parts of his body especially those on the paralyzed side, and objects around the room—pencil, clock, vase, shoe and the like With severe impairment, he will not recognize his hand as his own although he may recognize it as a hand; with moderate impairment he will name some articles but others will be missed In the mildest case, one will find ability to name an object as a whole but inability to name its parts, for example, the patient may be able to identify a wrist watch but not to name its stem, strap or hands.

Loss of *color* recognition can be brought out by asking the patient to name the color of different articles or to pick specific colors from an assorted group of cards. Color blindness must be excluded as a cause of failure.

Auditory Identification. Ask the patient with his eyes closed to identify such sounds as those made by jingling coins, crumpling paper, breaking a tongue depressor, or rubbing one's hands together or on a pillow. The response may vary from inability to recognize any sound to failure to recognize only those which are most complex or least familiar.

Tactile Identification. While the patient's eyes are closed, place in his hand one after another such articles as a coin, key, comb, and watch, and ask him to name them. If he cannot speak, one of these objects can be temporarily put in his hand and then laid on a table along with the others, he is then asked to point to the one which he held With less severe impairment, the patient may be able to identify an article but not its individual parts

Performance of Simple Acts. Inability to execute familiar purposive acts is known as *apraxia* As in writing, allowance must be made for weakness, paralysis, or other factor hampering muscular control Serious impairment may be indicated by inability to lace a shoe, tie a knot, or button his pajama coat If provided with a toothbrush, key, or cigarette lighter, the patient may be able to name it or even state its use but will fumble aimlessly when asked to put it to use. If the more simple procedures can be properly executed, apraxia may become evident if he is asked to perform a somewhat more complex test such as draw a house, bicycle, or a simple map of some familiar ground such as the route from his home to the near-by bus stop Deficiency in this field is most likely due to a lesion of the parietal lobe, precentral gyrus of the frontal lobe or, rarely, the occipital.

Sensory System

An accurate sensory examination requires time and perseverance Any factor interfering with cooperation, such as poor attention, low intelligence, language barrier, or diminished mental acuity, creates an added difficulty and renders the findings less reliable. *Comparison of corresponding parts of the body will, in the case of a unilateral lesion, provide the best control and make it possible to distinguish between normal and abnormal areas of sensation* In a bilateral lesion, one must rely on comparison of different levels of the body The patient's *eyes must be closed and, as an added precaution,* the examiner should occasionally go through the motion of applying the stimulus without actually so *doing.*

Minor or inconstant sensory impairment has no significance unless found with other more clear-cut signs of trouble

Tactile Sensation. The skin is touched as lightly as possible with a wisp of cotton or a soft camel's-hair brush Since pressure is distinct from touch sense, firm or even light pressure with the finger or hand must be avoided. In determining the correct minimal stimulus, the examiner is guided by his own experience and by first testing portions of the body where sensation is unimpaired In this, as in all sensory tests, allowance must be made for the normal difference in sensitiveness in various parts of the body

Superficial Pain Sensation. To discover whether superficial pain sensation is

lost (*analgesia*), diminished (*hypalgesia*), or increased (*hyperalgesia*), one pricks the skin with a sharp pin or needle A common straight pin is too dull. The required degree of pressure is determined by first testing unaffected parts. Analgesia or hypalgesia occurs in peripheral neuropathy, disorders of the spinothalamic tract such as cord tumor or injury, syringomyelia, and hysteria. Hyperalgesia is most commonly encountered in local disease or irritation of sensory fibers in the nerve or its posterior root.

Deep Pain Sensation. *Nerve* tenderness, elicited by pressing along the course of a nerve trunk, is found most often in peripheral neuropathy. *Muscle* tenderness is also present in neuropathy as well as in acute myositis. In testing for muscle or nerve pain, one must be careful not to confuse it with that due to thrombophlebitis, periosteitis, or other local lesion

In tabes dorsalis, the tenderness, which in the normal person can readily be elicited by squeezing the Achilles tendon, is diminished or absent

Temperature Sensation. Two test tubes, one containing cold water (15°–20°C) and the other, hot water (40°–50°C.) are the stimuli. Impairment of temperature sensitivity is almost always found over the same areas as altered pain sensation, in general, what applies to one applies to the other

Vibratory Sensation. The base of a vibrating tuning fork (about 128 vibrations per second) is applied over various bones, starting with the terminal phalanges of the upper and lower extremities The patient is requested to indicate if he feels the vibrations and when they appear to stop. The ability of the normal person to perceive them is dependent on a number of factors, including the type of fork used, mental alertness, and the thickness of tissues between skin and underlying bone Dependable standards can be learned only through experience In general, the normal person should clearly feel the vibrations for approximately 10 seconds If perception is absent or of short duration over the phalanges, one then performs the test at successively higher levels—the metacarpals, lower ends of radius and ulna, olecranon and achromial processes, metatarsals, malleoli, shin, patella, iliac crest and perhaps even the spinous processes Vibratory acuity is impaired along with all other sensations in peripheral nerve disease, obviously the higher it is impaired, the greater the nerve damage The chief value of the test is in estimating function of the posterior columns which convey vibratory and position sensation

Position Sense. The simplest procedure is to alternately flex and extend the large toe or index finger and have the patient, whose eyes must be closed, indicate the direction of movement The speed of motion must be constant The normal person will be able to detect the slightest change, especially of the finger In impaired position sense the digit must be moved through a wider arc before the patient can discern its direction; in the severe case he may be unaware of movement even to extreme positions This variant occurs under the same circumstances as diminished vibratory sensation although the two faculties may not be equally affected

When sense of position is appreciably impaired the finger-to-nose and heel-to-shin tests may also be poorly executed and, in general, voluntary movements are

not well coordinated (*sensory ataxia*). Here the action will be defective only when the patient is not permitted to watch his movement; if he does *watch it*, visual guidance will offset loss of position sense.

Position sense can also be tested by the *Romberg* procedure. Here the patient is asked to stand steady with his feet together, first with his eyes open, then closed If he is steady with them open but sways or loses balance with them closed, the test is positive. This is most likely to occur in peripheral neuropathy, [illegible] cerebellar [illegible] closed, although the unsteadiness is more pronounced with them closed. This is not a positive Romberg test. In psychoneurosis, one often notes swaying back and forth, reeling, or even falling, but there is less unsteadiness when the patient's attention is distracted by having him perform some other test such as the finger-to-nose, simultaneously with the Romberg

In any of the above procedures due allowance must be made for weakness, stiffness, or other variant hampering normal muscular activity

Stereognostic Sense. The patient is asked to identify in turn a number of familiar objects such as coins, keys, safety pins, or short pencils placed in his hand If touch sense is grossly impaired, the stereognostic tests are obviously of little value Astereognosis usually indicates a lesion in the parietal area of the contralateral side of the brain

Two-Point Discrimination. The stimulus is a small compass with blunted tips; one determines to what distance the tips must be separated before the patient can tell whether he is being touched by one or both. Two-point discrimination varies considerably in different parts of the body and in different persons, it is more acute in the fingertips, lips and face than elsewhere, and in the palms than in the soles The normal response in this test is so variable that definite impairment can be established only when it is possible to compare corresponding areas. Diminution of two-point discrimination has essentially the same significance as astereognosis.

Deep Reflexes

When a reflex is tested it is essential that the patient be relaxed, the pertinent part in proper position, and for accurate comparison of the two sides, the corresponding parts be symmetrically placed and the stimulus applied with equal force. Except where otherwise noted, a tendon (deep) reflex is tested by tapping the tendon with a reflex hammer.

The normal response is a single, momentary contraction of the muscles (jerk) to which the tendon is attached, followed by an immediate return to the previous status If no response is obtained, reinforcement should always be tried. For the legs, the customary procedure is to have the patient hook the flexed fingers of one hand into those of the other and pull while the examiner taps the tendons; for the arms, to have him clench his jaw or press downward with his heels on the table or floor Care must be taken not to confuse a reflex response with a voluntary or involuntary jerk due to pain or nervousness.

Different persons and the same person at different times vary so widely in their responses that it is not always easy to decide when a reflex is abnormal. What in one individual is merely a normal, brisk reaction may, in another, constitute a pathologically exaggerated response, in tense persons corresponding reflexes are often lively but sometimes the patient will hold himself so rigidly that it is difficult or impossible to elicit a reaction. As a general rule, if a reflex is pathologically hyperactive, other signs will be found in the same extremity such as clonus, Hoffman sign, or Babinski sign.

Inequality of reflexes, provided they are properly tested, usually means disease, but whether the more or the less lively is the abnormal reaction can often be determined only on the basis of other findings. A pathologically active tendon reflex usually indicates disease of the cortex or corticospinal tract; a pathologically diminished or absent one, peripheral nerve, anterior horn cell, or posterior column disease

Jaw Reflex. With the patient's mouth slightly open and jaw muscles relaxed, the examiner places his finger horizontally in the groove just above the point of the chin and taps it with the hammer Normally one finds no movement or a slight closing motion of the jaw A brisk reaction may be found in bilateral disease of the corticobulbar tracts, as in pseudobulbar palsy, amyotrophic lateral sclerosis or, sometimes, multiple sclerosis

Biceps Reflex. The patient's forearm, well-relaxed and partially flexed and pronated, is rested on the examiner's left forearm in such a way that the latter's thumb can be pressed lightly on the patient's biceps tendon. A hammer tap on the examiner's thumb, transmitted to this tendon, elicits contraction of the biceps muscle, resulting in a slight flexion movement of the forearm. An alternative method with the patient sitting is to have him rest his forearms partially flexed and with palms down on his homolateral thighs If he is supine, they can be partially flexed and rested palms down on the abdomen. In either case the examiner's thumb is placed on the tendon and tapped as just indicated As a rule, slight asymmetry can be better detected with the patient upright. Both the biceps and triceps responses are absent or barely perceptible in some normal persons

Triceps Reflex. The patient's arm is supported in the same fashion as for the biceps reflex, except that the triceps tendon must not be covered by the examiner's hand *Any of the positions just described can be used.* The hammer-tap is applied directly to the tendon just above the olecranon process. A slight extension movement of the forearm due to contraction of the triceps muscle is the normal reaction Another method is to support the patient's upper arm abducted to shoulder level with his elbow flexed and forearm and hand hanging vertically This has the merit of bringing out a posssible *pendular* response which is most likely to be found in cerebellar disease as a result of diminished muscle tone; instead of showing the customary single jerk following the hammer-tap the forearm will swing back and forth several times.

Radial Reflex. The patient's forearms can be rested on his thighs or abdomen, as in testing for the biceps jerk Or with his left hand the examiner can grasp



FIG 36 1 Testing biceps reflex

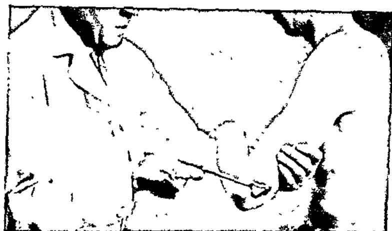


FIG 36 2 Testing triceps reflex

the patient's two forefingers and support them in front of the midline of the latter's body *in such a way that his upper arms hang loosely at his side*, his elbows are flexed to 90°, and his forearms slightly pronated The forearm is tapped just proximal to the styloid process of the radius, the normal response is slight supination and flexion movement of the hand

Finger Reflex (Finger Jerk). With his left hand the examiner supports the patient's hand in a relaxed position Between his own right fore- and mid-fingers he then grasps the patient's mid-finger in such a way that its metacarpo phalangeal joint is extended and the interphalangeals partially flexed. Next he snaps his right thumb off the edge of the patient's middle fingernail, thus causing the two distal phalanges to jerk into extension In most normal persons the thumb and free fingers will not move In some, especially those who are tense, they *will show a slight flexion motion, this is Hoffman's sign* When unilateral, this response is indicative of trouble, most likely in the corticospinal



Fig 36.3 Testing radial reflex



Fig 36.4 Testing finger reflex

tract Since a bilateral response is sometimes found in normal persons it can be regarded as significant only when other neurologic signs are detectable

Patellar Reflex (Knee Jerk). The patient sits forward on a chair with his legs partially flexed so that his feet are well forward and, although resting on the floor, bear no weight Or he can sit on the table with his legs hanging free over its edge provided his feet do not reach the floor If he is in bed, the examiner supports both extremities by placing his left forearm and hand under the knees; the thighs and legs should be slightly flexed with the heels resting lightly on the bed A hammer tap on the quadriceps tendon immediately below the patella causes contraction of the quadriceps group with resultant momentary



FIG 36 5 Testing patellar reflexes in ambulatory patient



FIG 36 6 Testing patellar reflexes in bed patient

extension of the leg at the knee. By placing one hand on the thigh the examiner can feel the muscles contract If the jerk is not obtained, reinforcement should be tried by having the patient interlock his fingers and pull those of one hand against those of the other In one who is supine, another method of reinforcement is to have him push his heels straight downward on the table Diminished or absent knee jerk is found in peripheral nerve, anterior horn cell, and posterior column disease, hyperactive jerk, in tense subjects and in disease of the corticospinal tract. When the knee jerks are unusually active, inequality can best be



FIG 36 7 Testing Achilles reflexes in ambulatory patient

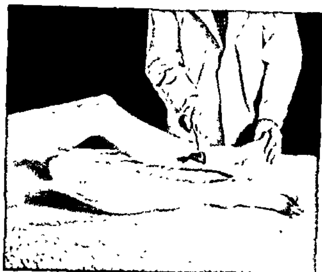
detected by tapping lightly first on one then on the other quadriceps tendon just above the patella, the response is an upward jerk of the latter.

In cerebellar disease the leg on the side of the lesion will, following excitation of the reflex, continue for a few moments to swing back and forth in pendular fashion (*pendular knee jerk*) Obviously, this can be brought out only if the patient is sitting on the table with his legs hanging free.

Achilles Reflex (Ankle Jerk). The patient kneels on a chair with his feet extending about 10 inches beyond its edge or sits on the table with his legs hanging free While holding the foot in slight dorsiflexion the examiner strikes the Achilles tendon, the response is a plantar-flexion movement of the foot. In a bed patient he can support the leg in slight flexion and hold the foot in slight dorsiflexion or have him flex his leg and thigh and evert his foot so as to make the Achilles tendon accessible If no response can be elicited by these methods, one should then test with the patient prone, his legs flexed to 90° and feet held steady by pressure of the examiner's left thumb and fingers on the balls of the feet

Pathologic ankle jerk has the same significance as pathologic knee jerk. It is important because in early peripheral neuropathy or in a lesion low in the cord or cauda equina, it may be abnormal, while the knee jerk is normal Diminution or absence of Achilles response on the affected side is of significance in the diagnosis of posterior dislocation of an intervertebral disc. Both ankle reflexes are usually absent in tabes dorsalis.

Patellar Clonus. With the leg extended, the patella, grasped between one's thumb and forefinger, is abruptly pushed downward and firm downward pressure momentarily maintained Normally there will be no response. Clonus is indicated by a series of rhythmical up and down jerking movements of the patella As a rule this is corroborative evidence of a pathologically active knee jerk, although rarely it is found bilaterally in a tense person.



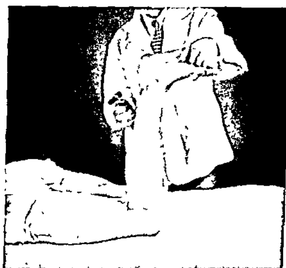


FIG 36 8 Testing Achilles reflexes in bed patient

Ankle Clonus. *With his knee extended, the patient's foot is briskly pushed into* dorsiflexion and held there. Normally there is no response, if a few clonic jerks of the foot are elicited, we have *unsustained* clonus; if clonic jerks continue as long as the foot is held in dorsiflexion, *sustained* ankle clonus If found unilaterally, either type indicates disease of the corticospinal tract. When found bilaterally, trouble can be assumed only if other signs of a corticospinal tract disturbance are detected.

Straight-Leg Raising. Although not a test of reflexes, straight-leg raising is included here because for convenience it is usually tested just before or after the examination of the reflexes of the lower extremities. It is important neurologically in bringing out irritation of the lower spinal nerve roots With the

patient supine the examiner, keeping the extremity straight, lifts it upward by the ankle, thus flexing it at the hip The other leg should be kept flat on the table. Normally, flexion to an angle of 70°–90° is possible although, especially in the older age group, allowance must sometimes be made for lack of general flexibility. If a lower nerve root is irritated, as in protruded intervertebral disc, meningitis, or subarachnoid hemorrhage, straight-leg raising by stretching the nerve and thus causing a pull on its root will create or accentuate pain in the lower back, buttock, or along the distribution of the sciatic nerve; flexion at the hip will be restricted by the pain. Discomfort in the thigh posteriorly due to stretching of the hamstring muscles is to be disregarded. Before impaired straight-leg raising man be attributed to root irritation, spasm of regional muscles by some musculo-ligamentous disturbance such as lumbosacral strain must be excluded.



Fig 369 Test for Kernig's sign.

Following straight-leg raising the thigh is again flexed, but with the leg flexed at the knee; since the nerve is not stretched, root irritation will not be accentuated If pain is increased or thigh flexion restricted, trouble in or around the hip joint is to be suspected. However, if with the thigh and knee both flexed, one then attempts to extend the lowe. l : stretching of the nerve will again accentuate root irritation and thus re·· novement (*Kernig's sign*)

If the results of these procedures are equ e has reason to suspect the presence of meningeal irritation, as in meningitis, nterior poliomyelitis, or subarachnoid hemorrhage, it is wise at this point to agai test for Brudzinski's neck sign If, with the patient supine, passive flexion of the head toward the chest induces flexion of the lower extremities at the hips and knees, meningeal irritation can be assumed

Superficial Reflexes

Corneal Reflex. *See above*

Pharyngeal Reflex. *See* Chap 1.

Abdominal Reflex. With the patient recumbent, the segment of skin overlying each abdominal quadrant is in turn briskly stroked with a sharp object such as the tip of a nail file or a paper clip. The normal response is momentary contraction of the abdominal muscles in the segment tested. Too sharp an object such as a pin cannot be used since by creating pain it is apt to cause a semivoluntary, defensive contraction of the entire abdominal musculature In a person with a fat or lax abdominal wall or a woman in late pregnancy an absent response is of no significance, otherwise a diminished or absent reaction suggests a lesion of the corticospinal tract This variant is regarded as an important early sign of multiple sclerosis.

Cremasteric Reflex. Here one strokes the inner aspect of the upper thigh The normal response is a brisk contraction of the homolateral cremasteric muscle, with resultant quick upward movement of the testicle It has the same significance as the abdominal reflex, being absent or diminished in disease of the corticospinal tract. Slow elevation of the testicle attributable to contraction of the smooth muscle fibers of the dartos layer is not significant. There is no corresponding test in females.

Plantar Reflex. The lateral aspect of the sole of the foot is stroked from the heel toward the fifth toe Here too, one must use a pointed object but not one so sharp as to cause pain and consequent voluntary withdrawal of the foot. In the normal person, all the toes show some degree of plantar flexion The reflex is abnormal when, instead of plantar flexion, there is extension (dorsiflexion) of the large toe (*Babinski's sign*); the remaining toes usually flex and may show slight spreading but their action is irrelevant Care must be taken to distinguish the reflex response from possible voluntary movement due to tickling or pain The Babinski sign almost invariably indicates a serious disorder of the nervous system, most likely a disturbance of the corticospinal tract

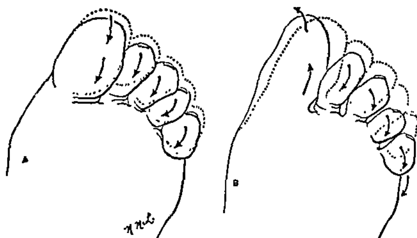


FIG. 36 10 Plantar reflex.

A. Normal Plantar flexion of all toes.
B. Abnor[illegible]nski sign) Extension (dorsiflexion) of great toe, plantar flexion of others.

or its cells of origin. Two exceptions to this rule should be noted: (1) The sign is a normal variant in infants too young to walk (2) Rarely, it is encountered in a lower motor neuron disturbance affecting the flexor muscles but sparing the extensors, as might happen, for example, in poliomyelitis; here the toes can extend but cannot flex No movement is obtained in peripheral neuropathy or anesthesia of the sole.

Other methods of eliciting responses comparable to those induced by plantar stimulation are:

1. Stroking downward along the median aspect of the tibia (*Oppenheim test*)
2. Stroking around the external malleolus and forward along the dorsolateral aspect of the foot (*Chaddock test*).
3. Squeezing the calf muscle (*Gordon test*).

These methods are particularly useful when the sole is anesthetic or so ticklish that one cannot properly evaluate the plantar reaction Of the three the Chaddock is the most reliable.

Autonomic Nervous System

The Eyes. Enophthalmos and exophthalmos, and the significance of pupillary changes have already been discussed (*see* Chap 4).

Vasomotor Changes. Vasomotor changes in the skin are of some significance in estimating the general autonomic status of the patient Ready blushing of the face, neck, and chest in response to stimuli which do not ordinarily affect most persons, or the rapid appearance of a red or white streak wherever the skin is lightly scratched, are indicative of emotional instability or an unstable autonomic system. Cold, clammy hands and feet and, when not attributable to overheating, sweating elsewhere, especially in the axillae, have the same significance. Anhidrosis occurs on the affected side of the face in superior cervical sympathetic paralysis (*see* Horner's syndrome, Chap 4) In disease affecting the autonomic nerves, such as Raynaud's syndrome, syringomyelia, or causalgia, the nails and soft tissues of the digits may be atrophic

Disorders of Urination. Involuntary micturition has already been discussed (*see* Chap 32) When not due to a local lesion of the genito urinary tract, it is most often encountered in states of impaired consciousness, mental disease, and spinal cord disease It may be one of the earliest signs of the last-named Precipitate micturition—inability to suppress the urge to void for even a few minutes—*occurs in lesions of the frontal lobe* In severe mental disturbances such as schizophrenia, and in deteriorative lesions such as general paresis and cerebral arteriosclerosis, the patient may void in public.

When bladder function is impaired as *a result of spinal cord* disease, the organ may be either atonic or spastic *Atonic* bladder may be created by impaired sensation with resultant lack of awareness of the need to void or develop in the early stage of an acute cord injury where damage to the *motor pathway* causes inability to perform the act *Spastic* bladder, most likely to be encountered in the later stages of bilateral corticospinal tract disease, causes frequent and involuntary urination When due to a lesion of the conus medullaris, *incontinence*

Abdominal Reflex. With the patient recumbent, the segment of skin overlying each abdominal quadrant is in turn briskly stroked with a sharp object such as the tip of a nail file or a paper clip The normal response is momentary contraction of the abdominal muscles in the segment tested. Too sharp an object such as a pin cannot be used since by creating pain it is apt to cause a semi-voluntary, defensive contraction of the entire abdominal musculature In a person with a fat or lax abdominal wall or a woman in late pregnancy an absent response is of no significance, otherwise a diminished or absent reaction suggests a lesion of the corticospinal tract This variant is regarded as an important early sign of multiple sclerosis

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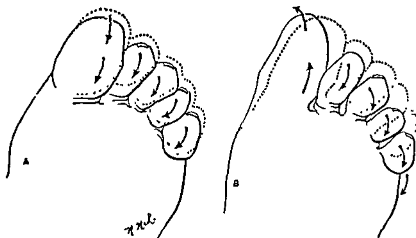


Fig 36 10 Plantar reflex

A Normal Plantar flexion of all toes

B. Abnormal (Babinski sign) Extension (dorsiflexion) of great toe, plantar flexion of others

Cauda Equina

Nerve roots involved within spinal canal. Manifestations usually bilateral but often asymmetrical

Impairment motor, sensory, or both. Sphincter disturbances frequent.

Present in. Tumor. Ruptured intervertebral disc

Spinal Cord

Always to be considered when any of the following features are present:

Manifestations bilateral, though often asymmetrical

Manifestations only below a certain level

Signs compatible with disease in one or more structures of the cord (*see below*)

Impairment, motor or sensory, *shows segmental distribution*

Disturbances of gait.

Paraplegia

Sphincter disturbances.

Anterior Horn Cells

Weakness or paralysis. Fascicular twitchings of degenerating muscles

Atrophy

Tendon reflexes diminished or absent

Distribution segmental

Sensation normal. Rapidly degenerating muscle painful and tender

Present in. Acute anterior poliomyelitis and progressive muscular atrophy (present in pure form). Compression, usually tumor, amyotrophic lateral sclerosis, syringomyelia (present in association with disease in other parts of the cord)

Corticospinal Tract

Weakness or paralysis. Increased tonus

Spasticity. *Contractures in long standing cases*

Atrophy absent or slight

Tendon reflexes increased

Plantar reflexes extensor in type (Babinski sign)

Abdominal reflexes diminished or absent

Present in. Multiple sclerosis. Subacute combined degeneration of the cord. Cord injury or tumor. Syringomyelia. Amyotrophic lateral sclerosis. Friedreich's ataxia. Acute myelitis. All of these usually show disease of one or more other parts of the cord. (Evidence of corticospinal tract disease also found in many cerebral and brain stem lesions.)

Posterior Columns

Ataxia. Romberg sign present

Tendon reflexes diminished or absent

Sensation. Vibration, position, and passive movement impaired or absent, tactile and pain sensation normal

Present in. Tabes dorsalis (present in pure form). Subacute *combined degeneration* of the cord, Friedreich's ataxia, compression or injury of the cord (present in *combination with disease in other parts of the cord*)

Spinothalamic Tract

Sensations of pain and temperature impaired or absent. (Although some of the tactile sensory fibers run in this tract, others follow a different pathway, *so that* we may have dissociated anesthesia. pain and temperature sense being impaired, while tactile sensation remains normal.)

Present in. Syringomyelia. Hematomyelia. Intramedullary tumor (rare)

Brain Stem

Motor or sensory disturbance in the regions supplied by one or more cranial *nerves*, excluding the first and second

Signs of corticospinal tract or of sensory disturbance *in extremities* and trunk on the contralateral side may be present.

or retention of urine (or feces) appears early; when due to a lesion of the cauda equina, late.

Disorders of Defecation. The neurologic disturbances which impair the urinary mechanism may also cause either inability to defecate, or fecal incontinence. Voluntary or involuntary passage of small amounts of liquid fecal material is often a reflection of impaction irrespective of its cause.

Anal Reflex. Contraction of the anal sphincter normally follows introduction of one's finger into the rectum. In tabes dorsalis or a traumatic, neoplastic, or inflammatory lesion of the cauda equina or conus medullaris, this reflex is absent; after withdrawal of the finger the sphincter remains relaxed.

Relation of Clinical Findings to Site and Character of the Lesion

The outline below indicates the important clinical findings in disturbances of the various parts of the nervous system and the more common diseases in which such disturbances occur.* It must be remembered that not all of the symptoms and signs characteristic of a particular disease are present in every case. For convenience, diseases of the muscles are included although they are not always due to changes in the nervous system.

Muscle
- Weakness
- Atrophy often present
- Tendon reflexes diminished or absent
- Sensation normal
- Involvement bilateral and more or less generalized. May be asymmetrical
- Present in: Myasthenia gravis. Progressive muscular dystrophy

Peripheral Nerves
- Weakness or paralysis
- Atrophy of muscles
- Tendon reflexes diminished or absent
- Paresthesia, pain, muscle tenderness
- Sensation impaired, all forms usually affected. (Subjective sensation such as numbness and tingling may be present when no sensory impairment can be objectively demonstrated.)
- Involvement, in focal lesions, corresponds to plexus or peripheral nerve distribution, in polyneuropathy, extremities symmetrically or asymmetrically involved, depending on the cause
- Present in: Injury of or pressure on a plexus or nerve. Tumor of or pressing on a nerve. Leprosy. Polyneuropathy

Nerve Roots
- Manifestations in general same as in peripheral nerve disease except that they conform to nerve root or segmental distribution
- Impairment motor, sensory, or both, depending on whether anterior, posterior, or both roots are involved
- Signs of root disturbance may be obscured by concomitant disease elsewhere (i.e., peripheral nerves or spinal cord)
- Present in: Cord compression (motor, sensory, or both). Ruptured intervertebral disc (motor, sensory, or both). Tabes dorsalis (sensory). Herpes zoster (sensory). Acute idiopathic polyneuropathy (chiefly motor)

*Prepared with the assistance of Dr. Charles S. Kubik.

Cauda Equina

Nerve roots involved within spinal canal Manifestations usually bilateral but often asymmetrical

Impairment motor, sensory, or both Sphincter disturbances frequent.

Present in Tumor Ruptured intervertebral disc

Spinal Cord

Always to be considered when any of the following features are present

Manifestations bilateral, though often asymmetrical

Manifestations only below a certain level

Signs compatible with disease in one or more structures of the cord (*see below*)

Impairment, motor or sensory shows segmental distribution

Disturbances of gait

Paraplegia

Sphincter disturbances

Anterior Horn Cells

Weakness or paralysis Fascicular twitchings of degenerating muscles

Atrophy

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Weakness or paralysis Increased tonus

Spasticity Contractures in long standing cases

Atrophy absent or slight

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Abdominal reflexes diminished or absent

Present in Multiple sclerosis Subacute combined degeneration of the cord Cord injury or tumor Syringomyelia Amyotrophic lateral sclerosis Friedreich's ataxia Acute myelitis All of these usually show disease of one or more other parts of the cord (Evidence of corticospinal tract disease also found in many cerebral and brain stem lesions)

Posterior Columns

Ataxia Romberg sign present

Tendon reflexes diminished or absent

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Spinothalamic Tract

Sensations of pain and temperature impaired or absent (Although some of the tactile sensory fibers run in this tract others follow a different pathway, so that we may have dissociated anesthesia pain and temperature sense being impaired, while tactile sensation remains normal)

Present in Syringomyelia Hematomyelia Intramedullary tumor (rare)

Brain Stem

Motor or sensory disturbance in the regions supplied by one or more cranial nerves, excluding the first and second

Signs of corticospinal tract or of sensory disturbance in extremities and trunk on the contralateral side may be present

Unsteadiness, intention tremor, nystagmus, nausea, perhaps Horner's syndrome
Present in Anterior poliomyelitis and progressive bulbar palsy (motor signs only) Medullary infarct Tumor Encephalitis *Multiple sclerosis* Acute idiopathic polyneuropathy Diphtheric neuropathy Syphilis Syringobulbia

Cerebellum

Incoordination (unsteadiness or ataxia, eyes open or shut)
Intention tremor
Nystagmus
Muscle tonus diminished (often)
Signs ipsilateral
No sensory impairment
Present in Tumor Multiple sclerosis Friedreich's ataxia and other heredo degenerative cerebellar diseases

Cerebrum

Personality changes, impairment of memory, confusion, disorientation, drowsiness, stupor, coma
Headache
Hemianopsia
Aphasia
Convulsive seizures
Corticospinal tract disturbances on contralateral side (hemiparesis or hemiplegia)
Stereognostic, position, passive movement, two point discrimination sense impaired on contralateral side Little or no demonstrable impairment of gross tactile, pain, temperature, and vibratory sensation, except in very extensive lesions In the thalamic syndrome (usually resulting from infarct in the thalamus) painful and other unpleasant sensations are more distressing than normal and spontaneous pain may be present on affected side of body
Present in Infarction Hemorrhage Tumor Abscess Injury

The manifestations of disease in the various parts of the nervous system as outlined above serve chiefly to localize the lesion, regardless of its cause The nature of the disturbance must often be determined by other clinical means.

FORM FOR NEUROLOGICAL EXAMINATION

Except for a few changes, the following outline is a reproduction of that employed on the Neurologic Service of the Massachusetts General Hospital It should be used only as a guide and perhaps as a check list for normal findings; abnormal findings must always be described in detail

Name **Date**

General. State of Consciousness Cooperation Intelligence

Cranium. Symmetry Size Tender? Bruit?

Spine: Head Posture Movements Stiff Neck? Brudzinski?
Scoliosis? Kyphosis? Lordosis? Tender?

Cranial Nerves: I Smell
II Visual Acuity Rt Lt Visual Fields Rt Lt
Fundi Rt Lt

III, IV, VI Movements Convergence Ptosis?
Diplopia? Nystagmus? Dysmetria?
Pupils Size Rt Lt Shape Rt Lt
Reaction to Light Rt Lt
Reaction to Accommodation Rt Lt
V Sensory Touch Pain Corneal Reflex
Motor Jaw Movements Jaw Reflex Muscle Nutrition
VII Facial Movements Volitional Emotional
Palpebral Fissure Naso labial Fold
Drooping of Mouth Corner? Rt Lt
Chvostek? Tremor?
VIII Hearing Air Conduction Rt Lt
Bone Conduction Rt Lt Vertigo?
IX Taste Subjective Objective (if indicated)
Sensation of Posterior Pharynx and Tongue
X Swallowing Palate Movement Pharyngeal Reflex Voice
XI Rotation of Chin Rt Lt
Elevation of Shoulder Rt Lt
XII Tongue Movements Deviation? Atrophy?
Fasciculation? Tremor?

Motor System General Gait Posture Articulation
Right or Left Handed Skilled Acts Tremors?
Fasciculation? Involuntary Movements?
Associated Movements Seizures?
Arms Posture Muscle Strength Nutrition
Coordination Finger to Nose Test
Past-Pointing? Dysmetria? Tremor?
Involuntary Movements? Adiadochokinesis?
Decomposition of Movement Rebound Phenomenon?
Legs Posture Muscle Strength Nutrition
Coordination Dysmetria? Heel to Shin Test Tremor?
Involuntary Movements? Rebound Phenomenon?
Straight Leg Raising Kernig?
Trunk Muscles Shoulder Girdle Thorax
Abdomen Pelvic Girdle

Language and
Related Functions Spoken Words Performance Understanding
Written and Printed Words Performance Understanding
Identification Visual Auditory Tactile
Performance of Simple Acts

Sensory System Arms Tactile Superficial Pain Deep Pain
Temperature Vibratory Position Sense
Stereognosis Two point Discrimination
Legs Tactile Superficial Pain Deep Pain
Temperature Vibratory Position Sense
Two point Discrimination Romberg
Achilles Tendon Sensitivity
Trunk Tactile Superficial Pain Deep Pain
Temperature Vibratory
Two point Discrimination

Deep Reflexes	Right	=	Left
Jaw (*see Cranial Nerve V*)			
Biceps			
Triceps			
Radial			
Finger			
Patellar			
Achilles			
Patellar Clonus			
Ankle Clonus			

Superficial Reflexes	Right	=	Left
Corneal (*see Cranial Nerve V*)			
Pharyngeal (*see Cranial Nerve X*)			
Upper Abdominal			
Lower Abdominal			
Cremasteric			
Plantar			
Oppenheim			
Chaddock			
Gordon			

Autonomic System: Ex- or Enophthalmos? Pupils (*see Cranial Nerve III*) Salivation? Temperature of Extremities Nails Sweating? Skin Stroke Test Flushing? Heart Rate Sinus Arrythmia? Carotid Sinus Pressure Blood Pressure Lying Standing Disorder of Urination? Disorder of defecation? Anal Reflex

Endocrine System: Body Type Fat Distribution Hair Distribution Pigmentation Skull Jaw Thyroid Breasts Skeleton Long Bones Hands Sexual Development External Genitals Prostate Uterus

Other Abnormal Findings

Recapitulation of Important Variants

MENTAL STATUS

No neurologic examination is complete without a survey of the patient's mental status Observation made during routine history-taking and physical examination is in some cases insufficient, to avoid overlooking a serious mental defect, a more thorough investigation of the mental status may be necessary. For example, an old lady with diffuse changes in the brain who seems to carry on a normal conversation may on careful questioning be discovered to have a defective memory, especially for recent events, and to lack ability to reason, calculate, or plan Another patient, apparently normal, might on carefully directed questioning prove to be definitely paranoid

As in history-taking, the physician must have a natural, friendly, unstudied manner and carry on the interview as though it were a conversation instead of an examination. When it is left until the last, the patient, less ill-at-ease, will probably be more communicative However, if he appears fatigued by what has already transpired, postponement to the next day is advisable.

Appearance

Slovenliness is characteristic of the patient with schizophrenia, the drug addict, and the acute or deteriorated alcoholic; overdressed "flashiness" bespeaks

the manic; predilection for "health" belts, girdles, or braces suggests hypochondriasis In the facial expression, the practiced eye may recognize the confident smile of the manic, the melancholic mien of the depressed, the odd, mirthless, detached smile of the schizophrenic, the dull expression of the patient with brain tumor, the suspicious side-glances of the paranoid, the tenseness of the person with anxiety neurosis, and the frightened look of the delirious.

Attitude

The schizophrenic patient seems preoccupied and out of touch with his surroundings, the one with brain injury or tumor may appear apathetic but is easily pleased or angered; the manic is overfriendly, but once his moves or plans are opposed may become antagonistic and even violent The depressed person seems uninterested but may indicate by a nod that he understands what is going on. The delirious or stuporous patient is incompletely aware of his surroundings

Behavior

The manic is overactive and easily distracted by some superficial stimulus such as jingling of keys or coins Or he may break off in the midst of a conversation to comment on some irrelevant subject The schizophrenic is absorbed in his thoughts, difficult to distract, and his movements are stereotyped. The depressed patient is retarded, preoccupied and not easily distracted The agitated depressed person paces the floor, rubs his hands together and mutteringly reiterates some such phrase as, 'Oh my God!" The patient with hallucinations may be observed to nod his head, glance to the side, or mumble as if in communication with someone invisible to the examiner Ritualized or repeated motions such as touching or holding objects, or "picking" mannerisms bespeak compulsion neurosis

Stream of Talk

The examiner should always let the patient tell his own story, interrupting only to encourage or keep him to the point. Overtalkativeness is characteristic of manic, hypomanic, compulsive, delirious, and intoxicated persons The manic may make remarks which appear to be irrelevant, but which represent his attempt to pun or joke; flight of ideas is also evident. Depressed, schizophrenic or shy persons are taciturn and frequently show *blocking*—that is, stopping in the middle of a sentence, failing to reply to a question, or looking at the floor or uneasily across the room and showing no indication of resuming the conversation When intimately personal topics are touched upon most persons show *hesitancy* in talking In schizophrenia, it occurs regardless of the topic, the patient perhaps responding to some imagined command abjuring him from further talk The hypochondriac continually reverts to his own illness. Patients with neurosis change the subject when it approaches too closely to disturbing personal matters In schizophrenia, general paresis, and local or diffuse brain damage, speech may be a meaningless jargon

Mood

Although appearance and behavior give some indication of general emotional status, particular though guarded interrogation is often necessary Leading questions are to be avoided whenever possible. Queries such as "How do you feel?" or "Has any change in feelings come over you lately?" are more fruitful than such direct ones as "Are you depressed?" The depressed person is likely to be most downcast in the morning but feels brighter as the day goes on He complains of lack of energy, retardation of his thinking processes, and poor memory, but actual tests will show memory to be unimpaired

Every depressed patient is a suicidal risk and his feelings concerning suicide should be questioned, first indirectly, then if necessary, outright He may attempt dissimulation, professing to feel in the best of spirits when actually contemplating self-destruction In fact, any patient, who, contrary to reports from his family, vigorously denies being depressed, should be strongly suspected of suicidal tendencies. One who has made definite plans or purchased a gun or poison is in general a more serious risk than one who has only fleeting thoughts or fears of suicide *There is no foundation in fact for the widespread belief that the person who threatens suicide will never commit it* The manic or paretic patient feels full of energy and, unless crossed, is good humored Other suspect moods are those of perplexity, uneasiness, suspiciousness, apprehensiveness, and unreality.

Special Preoccupations

These may appear during a routine interview or be elicited only by careful interrogation

Delusion. This is a false belief or wrong judgment. For example, the patient may harbor the notion that he is being persecuted or that he has unlimited funds Paranoid delusions are particularly likely to center around radio, electricity, hypnotism, detectives, the F B. I., or persons of another religious faith When paranoia is suspected, such questions should be asked as "Are you being discriminated against?" "Do people talk about you behind your back?" Delusions of persecution occur in paranoid-schizophrenia, chronic alcoholism, and toxic psychosis Delusions of grandeur occur in general paresis and manic psychosis.

Hallucination. The patient possesses a false perception which lacks an external stimulus If he hears non-existent voices or sees imaginary objects, detailed questioning is necessary to distinguish between a hallucination and an actual noise or sight which is misinterpreted In schizophrenia, hallucinations are elaborate; well-defined voices and bizarre scenes and visions as of heaven, angels, or perhaps torture machines, are common In depressed states, the patient hears the voice of his conscience, condemning him for imagined short comings or wrong doings

Illusion. An external stimulus which actually exists is misinterpreted The patient hears the voice of a nurse but believes it to be that of his mother, or he might mistake the chair in his room for an animal, perhaps a ferocious one. In

toxic delirium associated with drug intoxication, the postoperative state, infectious disease, cardiac failure, or other disorder, one is likely to attribute a fearful import to ordinary occurrences

O[illegible] This is an idea of which a person cannot rid himself even though he [illegible] that he has neglected [illegible] may be found in normal children and adults

Compulsion. One feels compelled to perform some unreasonable or unnecessary action such as repeatedly washing his hands, touching certain objects, going back to see if the lights are off, or dressing and undressing in a ritualistic manner Compulsions occur in compulsion neurosis and depression. Normal children sometimes show compulsion of lesser import such as stepping over sidewalk lines or touching fence posts while walking down the street.

Phobia. The patient is beset by an unreasonable fear. It may be an accentuation of a normal one as of snakes, germs, knives, or high places, or be an entirely groundless fright as of drinking milk, meeting a dark-skinned man, being left alone, or crossing the street alone Characteristic of anxiety neurosis is the need to avoid crowds, one's church, stores, theaters, elevators, subways and any crowded or small "shut in" place, such dreads often seriously hamper ordinary social and business activities.

Sensorium

Orientation, memory, general information, reasoning, calculation, attention, judgment, and insight are affected in temporary severe disturbance of brain function as in toxic psychosis, and in permanent cortical damage such as that attributable to general paresis, arteriosclerosis, or tumor.

Orientation. Loss of awareness in relation to time, place, or personality is sometimes obvious but may be detected only by asking the patient to name the day of the week or month, the year and season of the year, the place of examination, the identity of the examiner, and so on. Disorientation occurs in toxic psychosis and brain damage from any cause Pseudo-disorientation may be encountered in a depressed patient, his characteristic reply to queries being "I don't know anything—not even the date" Here further questioning will show that this reply indicates loss of self-confidence or interest rather than of orientation

Remote Memory. The patient is asked for a chronological account of his life, with dates of birth, marriage, birth of children, and ages and names of his relatives Discrepancies, of which he is either unaware or unconcerned, are significant In general paresis, for example, although reporting that he is 50 years old, he may date his birth only 20 years back and be totally unconcerned over the obvious incompatibility of the two statements. Remote memory is impaired in cerebral arteriosclerosis, pellagra, general paresis and brain tumor, in the first named it is better than recent memory

Recent Memory. This is tested by seeking an account of the patient's experiences during the past few days "What time did you come to the hospital?"

"How did you get here?" "With whom did you come?" "What did you have for breakfast?" In senility, toxic psychosis, Korsakoff's syndrome, stupor from any cause, and amnesia, the patient remembers little or nothing of such recent events.

Immediate Recall. Here one tries to have the patient, immediately or within a few minutes, repeat digits, phrases, or a story just told to him When preoccupied, as in schizophrenia, he will be incapable of so doing because of lack of concentration on the stimulus The manic will fail because he is too distractible, some association sending him off on an irrelevant topic. Immediate recall is also lost in Korsakoff's syndrome and sometimes in senile psychosis

Calculation. Simple problems of addition and subtraction are first posed. If these can be correctly performed the "seven-from-one hundred test" can be tried; the patient is asked to begin with one hundred and continue to subtract seven until he reaches two These are actually more than tests for calculation; in addition to showing the degree of impairment they will suggest the nature of the psychosis The depressed patient may be too preoccupied to try or may perform very slowly, the manic will do the seven-from-one hundred test rapidly, perhaps jumping ahead by subtracting multiples of seven, the paretic will either not notice or not care about the mistakes he makes, the feeble-minded becomes bewildered, the neurotic may state that he is suffering too much to try.

General Information. This is estimated by asking for the names and election years of recent presidents, the largest cities in the country, dates of great wars, the name of the governor, mayor, or other local dignitary, names of state and foreign capitals Background and education must be taken into consideration; what one seeks is not the amount of information but a discrepancy between what the patient should reasonably be expected to know and what he actually seems to know

Judgment. Inquiries are made concerning business plans, social activities, family responsibilities, and ethical standards Loss of judgment is one of the earliest signs of cortical destruction, occurring in general paresis, brain tumor, and cerebral arteriosclerosis.

Insight. Here one evaluates the patient's reaction to questions about the nature of his illness and the proper course to be pursued for his benefit The neurotic may realize that nervousness is an important feature of his sickness; the more severe hypochondriac insists that all the trouble stems from his bowel, heart or other organ, or some hidden infection or malignant tumor The person with phobias, compulsions, or obsessions is usually aware that they are abnormal mental symptoms. In the early stages of schizophrenia, the patient may have a most distressing type of insight in which he feels he is "going crazy" and begs the doctor for help; otherwise he has poor insight. The manic is not likely to admit that anything is wrong and feels that his critics are obstructing his plans. The depressed often realizes that he is suffering from a depression and may remember previous episodes of a similar nature. In toxic psychosis there are intervals during which the patient realizes he is or has been confused

and may recognize that his hallucinations and delusions have no basis in fact. In paresis, there is no insight

Intelligence Tests

More or less quantitative determinations of a subject's ability to learn are made by giving intelligence tests The *Binet-Simon* and the *Bellevue* tests are commonly used, their description is outside the scope of this book.

LUMBAR PUNCTURE

Although examination of the spinal fluid is not within the scope of this book, it is appropriate to describe the technique of lumbar puncture. This procedure should always be performed where it will aid in diagnosis or treatment It should be avoided if possible in the presence of cutaneous or bone infection at or near the site of the puncture If elevated spinal fluid pressure is likely, as in high-grade choked disc or suspected brain tumor or subarachnoid hemorrhage, it should be performed with the greatest care because of the danger of death from "*compression cone*", here the needle must be of small caliber to facilitate slow removal of fluid and lessen the chance of persistent leakage through the hole in the arachnoid or dura following its withdrawal

Technique. The patient is placed on his side at the edge of the table or a firm bed The spine must be absolutely horizontal and the plane of the back perpendicular to the table. To widen the intervertebral spaces, the head, thighs, and legs are flexed Constant watchfulness must be exercised to make sure that this position is maintained, otherwise, the needle will be misdirected and the tap unsuccessful To minimize the danger of breaking the needle, the patient must be warned against any movement of his back throughout the procedure.

Puncture is usually performed between the third and fourth or fourth and fifth lumbar vertebrae Their spinous processes should be carefully identified, extra time so spent may spare the patient the pain of later probing When the site for insertion of the needle—in mid-line mid-way between the spinous processes of the vertebrae—is located, it is marked by a skin impression made by one's thumbnail Following proper sterilization, the skin and underlying tissue in the anticipated path of the needle are novocainized, and at least 4 minutes allowed to elapse for thorough anesthetization

The needle is now inserted through the skin at the point previously marked and directed perpendicularly to the back but aimed slightly toward the head It is pushed steadily forward until a sudden give is felt this signifies that the point has penetrated the ligamentum flavum and dura, and entered the lumbar sac If the stilette is now withdrawn, spinal fluid should appear If not, the stilette should be reinserted, the needle turned slightly, and the stilette again withdrawn If fluid still fails to appear, if the needle seems to have encountered bone, or its easy passage to have been otherwise obstructed, further manipulation will cause more pain or, if undue force is used, break the needle. Instead one should withdraw the needle to the skin level where it is freely movable,

check the position of the patient, and begin again. When difficulty is encountered in one space, it is sometimes wiser to try the one above Once the subarachnoid space is successfully punctured, the stilette is removed and the stopcock immediately turned to a neutral position to prevent loss of fluid and erroneously low pressure readings.

Measurement of Pressure. The manometer is now attached, the stopcock opened, and the initial pressure reading taken Normal figures, which are subject to considerable variation, average 130–150 mm. of spinal fluid, expressed as millimeters of water. When a reading is high, an interval of not less than 20 minutes should be allowed to pass before accepting it as correct, often, excitement or poor relaxation causes temporary elevation which will subside in the allotted time. If there is true elevated intracranial pressure, as in brain tumor, the figure will remain high. After a reading has been accepted as correct, the patient is instructed to cough or his abdomen is compressed by an assistant Normally, the fluid in the manometer will rise promptly, thus indicating free communication between the needle and the lumbar sac

If spinal block or lateral sinus or jugular bulb thrombosis is suspected, jugular compression is in order; otherwise it should be omitted since it might cause hazardous alterations of pressure relationships within the cranium in such a disturbance as brain tumor or hemorrhage To test for spinal block one compresses both jugular veins, the normal response is rapid rise to 300 mm or above Slow or negligible rise indicates subarachnoid block. Compression is maintained only long enough to determine the rate of change but for not more than 10 seconds, following release pressure will normally return promptly to its original level but will be slow or negligible in the presence of block Comparison of response to jugular compression on the two sides is helpful in establishing the diagnosis of jugular bulb or lateral sinus thrombosis but is otherwise of no importance If thrombosis is present, pressure rise will be absent or delayed on the affected side, prompt on the normal Before attributing such a difference in pressure changes to lateral sinus thrombosis one must exclude by x-ray any appreciable difference in caliber of the two sigmoid sinuses, an unimportant anomaly which will cause the same phenomenon.

Withdrawal of Fluid. Upon completion of pressure observations, spinal fluid samples are collected in three or four tubes (usually 3–5 cc in each) for distribution to the appropriate laboratories

When the pressure is elevated, fluid must be withdrawn slowly over a period of perhaps 30–60 minutes until the former has dropped to approximately one-half of its initial level It is believed that by so doing one greatly lessens the chance of creating a fatal "compression cone". As the fluid is withdrawn, readings are taken after removal of each 5 cc A large fall following release of a small amount points to a small reservoir, this occurs in expanding lesions of the cerebral hemispheres which encroach on the ventricles. A slight drop following removal of a large quantity indicates a large reservoir, as in hydrocephalus

The needle is now quickly withdrawn and the skin covered with a small dressing

Appearance of Fluid. Grossly bloody fluid may be accounted for by trauma during the puncture ("bloody tap"), or by blood already present in the fluid. In a bloody tap the amount of blood varies in the different tubes, usually diminishing as more fluid is withdrawn; in a centrifuged specimen, the supernatent fluid is colorless When blood is due to subarachnoid or cerebral hemorrhage it is evenly distributed in the various tubes, and except following recent bleeding, the supernatent fluid is xanthochromic. This yellowish discoloration is found in compression of the cord from any cause, in subarachnoid hemorrhage after a few days, and occasionally in acute idiopathic polyneuropathy. Cloudiness indicates a decidedly increased cell count. In acute meningitis the fluid may be frankly purulent.

37

DISEASES OF THE NERVOUS SYSTEM

In this chapter are discussed the more common disorders which create abnormalities of sensation and muscular activity (neurologic) and of perception, thinking, emotion, and behavior (psychiatric). As far as possible they are grouped on an anatomic basis.

THE MYOPATHIES

Myasthenia Gravis

Occurring at any age, but most often between 15–25 and 45–70, myasthenia gravis is due to an abnormality of the myoneural junctions There is evidence that it may be related to tumor or hyperplasia of the thymus gland It is characterized by abnormal fatigability and, sometimes, total impairment of movement of certain muscles, especially those supplied by the cranial nerves The process may be asymmetrical Dysphagia, dysarthria, difficulty in chewing, drooping of eyelids, diplopia or other indication of impaired ocular motor movements, and lack of facial expression are usually the first manifestations On swallowing, choking, regurgitation of fluid through the nose, or inspiration of food particles is likely In time, other muscles become affected, especially those of the neck, back, or respiration, and the proximal groups of the extremities, particularly the upper. The patient may eventually become unable to hold up his head, sit erect, or use his arms or legs. Deep reflexes are normal or slightly diminished, but become easily exhausted on repeated stimulation.

Most characteristic and of special value in diagnosis of the early case is the fatigability of a muscle or group of muscles brought out by repetitive use If instructed to perform a certain movement repeatedly, the patient will soon tire and be forced to stop, after a short rest he will be able to resume the same movement but again, only briefly

Diagnosis can usually be confirmed by creation of dramatic temporary improvement following intramuscular injection of 1.5 mg of neostigmine methylsulfate Virtually normal activity of affected muscles will be restored for the time being Before this test is undertaken other neurologic disturbances, especially progressive bulbar palsy, must be excluded, since the drug might aggravate paralysis already present and in so doing even cause death.

Remissions and relapses in varying intervals—weeks to months—are the rule. Many patients can be maintained for a long time in relatively good health by



Fig 37 1 Myasthenia gravis

A Before treatment Patient is trying to smile Note drooping of both lids, lack of expression, and weakness of facial muscles
B After treatment

adequate therapy with neostigmine but ultimate death from respiratory paralysis is likely.

Progressive Muscular Dystrophy

Although not actually a disease of the nervous system, progressive muscular dystrophy is included here for convenience Often familial and predominant in males, it begins early in childhood and is of long duration. It is characterized by a combination of atrophy of one group of muscles with pseudohypertrophy of others. The typical case shows atrophy of the shoulder, pelvic, thigh, and spinal extensor muscles, and pseudohypertrophy of the calves Prominent features are waddling gait, extreme lordosis of the spine with protrusion of the abdomen, and compensatory backward slant of the trunk When asked to stand from a supine position on the floor, the patient will turn over, push himself up on all fours, and then raise himself erect by gradually working his hands upward along his thighs Reflexes are normal or diminished Biopsy of an affected muscle shows a characteristic degeneration of its fibers In some cases cardiac hypertrophy and eventual failure develop from involvement of the cardiac muscle by a comparable process An unusually rapid course or appearance of symptoms in midlife or later should alert one to the possibility of dermatomyositis even though skin lesions are lacking

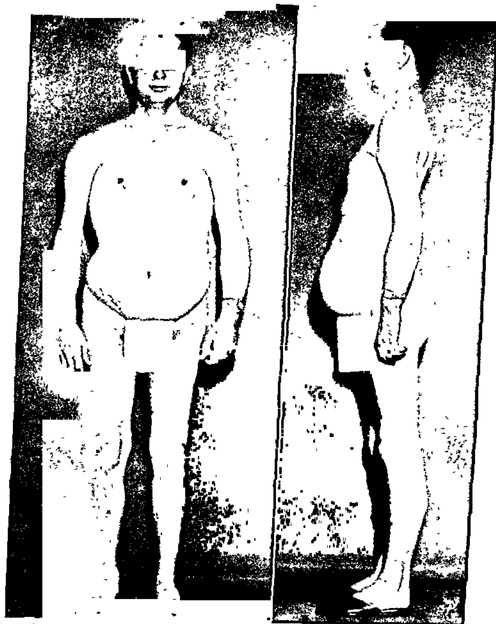


FIG 37 2 Progressive muscular dystrophy Atrophy of muscles of shoulder girdle, upper arms, and thighs Pronounced lordosis and prominence of abdomen due to weakness of back and abdominal muscles (Courtesy Dr Charles S Kubik)

THE PERIPHERAL NERVES

Clinically, neuropathy is defined as a usually, but not necessarily, painful degenerative process in any part of a peripheral nerve, creating functional impairment which, according to the anatomic part involved, is sensory, motor, or mixed Among its causes are local injury, deficiency, diphtheria toxin, certain

exogenous chemical toxins, and certain metabolic and vascular disturbances. In general, each cause tends to create a distinctive pattern with respect to the parts involved and the predominance of motor or sensory impairment, but this is by no means a fixed rule One may find decided individual variations in cases with the same etiologic background or similarities in those due to different causes When a nerve is gradually impaired, the symptoms and signs begin peripherally and progress proximally; the fingers are involved earlier than the hand and forearm Furthermore, in any generalized disturbance such as acute idiopathic polyneuropathy the distal parts supplied by the longer nerves are likely to be affected first; trouble will be found earlier in the toes than in the fingers Although paresthesia or pain is a frequent symptom of neuropathy, it must be emphasized that neither, even when occurring in a circumscribed area, is sufficient to justify this diagnosis, other signs of trouble must be detected For example, pain in the thigh might be a reflection not of neuropathy but of some other disturbance such as sacroiliac strain or spasm of low-back muscles However, one can be reasonably certain that a disease process has affected a peripheral nerve or a root if pain or paresthesia is localized to the distribution of one or the other and is accompanied by any local objective sensory change, muscular weakness, or tendon reflex impairment Only the more commonly encountered forms of neuropathy are discussed

Traumatic Neuropathy

Due to direct injury, tear, or stretch of a nerve, contracted scar tissue from previous injury, misdirected injection of a therapeutic agent, prolonged pressure as from a bony outgrowth or some external object, or pressure or invasion by tumor, traumatic neuropathy as a rule is unilateral and localized Depending on the nerve involved, the manifestations will be predominantly sensory or motor The patient may complain of pain or paresthesia in an area conforming to the anatomical distribution of a nerve One may find regional sensory impairment, muscular weakness, or both, and, if the process is severe or prolonged, muscular atrophy, paralysis, and diminished to absent tendon reflexes Vasomotor and trophic disturbances are likely when a sensory or mixed nerve is involved Pressure from a cervical rib causes paresthesia or dull pain in the arm followed by weakness, and in the hand and fingers, vasomotor changes, atrophy, and paralysis Localized neuropathy from injury to the brachial plexus is encountered in infants due to birth trauma and in adults as a result of violence The distribution of signs depends on which trunks are affected Injury to a nerve from prolonged pressure or interference with local circulation due to abnormal position of an extremity is frequently seen following stupor or coma For example, the patient's arm may have hung over the back of a chair throughout the night, he was unaware of the discomfort signal which would have impelled the non-stuporous person to change position Alcohol or drug intoxication and careless positioning of limbs during or after administration of surgical anesthesia are likely causes The musculospiral, ulnar, and peroneal nerves are the ones most vulnerable. Sometimes one finds gradual weakness and sensory

changes in the ulnar distribution due to small caliber of the groove through which the nerve passes over the elbow. Occasionally, a median nerve disturbance is a reflection of its compression by the annular ligament of the wrist; the acroparesthesias experienced by some persons, especially during the night, may well represent a mild form of this phenomenon. Trouble in the upper extremity attributable to neuropathy can also be a reflection of the scalenus anticus syndrome, cervical strain or arthritis, ruptured cervical disc, or pressure or invasion by tumor; in the lower extremity, of strain or arthritis of the low back, ruptured lumbar disc, or tumor

Causalgia

This is a hyperesthetic neurovascular syndrome occasionally encountered following injury or amputation The median nerve, the sciatic, or a branch of either is most often affected One finds moderate to agonizing, constant or paroxysmal pain in the palm, sole, or a digit, and extreme hyperesthesia sometimes so severe that the slightest touch is unbearable The skin on the affected part may be glossy, red, and warm, or cold and pallid; when a digit is involved, atrophic changes of the nail are likely. It is believed that injury to the sympathetic fibers in the nerve trunk is responsible

Meralgia Paresthetica

See Chap. 31

Vitamin Deficiency

The diet may be actually deficient in vitamins, or relatively so because of increased requirement, as in hyperthyroidism, pregnancy, or lactation Prolonged vomiting, gastritis, intractable diarrhea, or a surgical procedure such as gastrectomy or a short-circuiting operation sometimes prevents proper absorption Although it is often possible to identify a specific syndrome such as pellagra, scurvy, or beriberi, the line of demarcation between the various forms is not always clear-cut since shortage of one element is likely to be associated with shortage of others

Peripheral neuropathy is attributable to thiamine deficiency (*beriberi*) The patient complains of gradually increasing paresthesia or pain and fatigability of his extremities Weakness on walking or use of the arms soon becomes evident; bilateral foot drop with steppage gait is common Sometimes one finds complete flaccid paraplegia Tendon reflexes are diminished or absent All forms of sensation are impaired Tenderness of muscles or along the nerve trunks is the rule In addition to the signs of neuropathy one is also likely to find extensive peripheral edema and other signs of cardiac insufficiency (*see* Chap 22) Signs attributable to deficiency of other elements, such as pellagrous skin or mucous membrane lesions, hemorrhages due to scurvy, or corneal or cutaneous changes due to avitaminosis A may also be evident The spinal fluid is usually normal in contrast to many of the other forms of polyneuropathy in which a high protein content is the rule.

So-called *alcoholic neuritis*, a form of polyneuropathy found in the chronic excessive drinker, is attributable to deficiency of vitamins and other nutritional elements resulting from inadequate diet or impaired absorption of food. It is *not* a reflection of direct toxic action of alcohol. The clinical picture is identical to that just described. *Wernicke-Korsakoff syndrome*, also due to malnutrition from chronic alcoholism or other cause, is discussed under Alcoholic Psychosis (*see below*).

Herpes Zoster

The sensory spinal ganglia or their homologues in the cranial nerves are inflamed by a filtrable virus Mild or severe burning or cutting pain and hyperesthesia pursuing definite root distribution occur first After a few days the skin in parts of the same area becomes reddened and shortly thereafter shows clumps of vesicles which remain discrete or coalesce After a week or more the vesicles dry and scab over; the scab later desquamates, leaving a pigmented scar which may eventually disappear or, in the severe case, is sometimes permanent. The lower thorax and upper abdomen are the most common sites, but the head or an extremity may be the site Since the disease is almost invariably *unilateral*, such disturbances as pleuritis, pneumonia, acute cholecystitis, appendicitis or lumbosacral strain may be wrongly diagnosed before the skin lesions develop. Especially in older persons when the ophthalmic or intercostal nerves are involved, sharp, shooting pains and cutaneous hyperesthesia can persist for months or years With involvement of the fifth nerve ganglion, corneal ulcer or panophthalmitis may develop and perhaps end in permanent impairment of vision Although as a rule herpes zoster occurs independently, it is sometimes seen as a complication of a systemic disease such as acute meningitis, tuberculosis, Hodgkin's disease or leukemia, or local disease of the spine such as metastatic carcinoma or trauma Furthermore it is thought that the virus is in some way related to that of chicken pox since herpes zoster is not uncommon in persons recently exposed to the former but who give a history of having had it in the past

Diphtheric Neuropathy

Respiratory Diphtheria. Within 2–3 weeks after onset, respiratory diphtheria may be complicated by palatal paralysis, visual difficulty caused by paresis of the accommodation mechanism or ocular motor muscles, lid ptosis, or hoarseness or aphonia resulting from paralysis of vocal cords After 5–7 weeks the severe case can develop weakness or paralysis of one or more extremities, most likely the legs The regional tendon reflexes become impaired. Although the process is primarily motor, one occasionally finds paresthesias or glove or stocking type of hypesthesia, and nerve and muscle tenderness. The spinal fluid protein is elevated The picture is often indistinguishable clinically and by spinal fluid examination from that of acute idiopathic polyneuropathy; early involvement of cranial nerves favors diphtheria. Although the patient may die of respiratory paralysis, diphtheric myocarditis, or other complication, gradual improvement is the rule but recovery is rarely complete before several months.

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in the blood, the most important of which is stippling. Lead encephalopathy is characterized by headache, loss of memory, aphasia, pareses, convulsions, and in acute cases, by profound delirium, mania, or coma.

Acute Idiopathic Polyneuropathy

Also known as *infectious polyneuropathy* and *Guillain-Barré syndrome*, this disease is ushered in by acute or subacute fever, anorexia, and malaise. After a few days the patient develops paresthesia and weakness in the distal part of one or more extremities. As a rule, the toes and feet are involved first, the fingers and hands a day or so later The process spreads rapidly up the limbs, perhaps to the point of creating complete paralysis of all four extremities, the trunk, and many of the muscles of the neck and head Sometimes unilateral or bilateral weakness of the facial muscles appears before generalized involvement Pain, paresthesia, and objective sensory changes as a rule are transient and rarely striking In fact, they may have already disappeared by the time the patient is first examined Physical examination shows weakness to complete paralysis of one or more muscle groups, nerve or muscle tenderness, and diminished to absent tendon reflexes. Sphincter disturbances may occur. The spinal fluid shows increase of protein but normal cell count. Slow but eventual complete recovery is the rule. Respiratory paralysis is a hazard As noted above, to distinguish acute idiopathic polyneuropathy from diphtheric polyneuropathy is often impossible

Diabetic Neuropathy

A common form of neuropathy, this is most likely to occur in older patients, especially those whose diabetes has been poorly controlled and who have lost appreciable weight. The process is usually mixed, with sensory impairment predominating Asymmetrical involvement is the rule One or more extremities, most likely the lower, will first develop paresthesia or pain; the latter is sometimes severe. Mild to moderate objective sensory changes and often weakness and loss of deep reflexes soon appear. The muscles and nerves become tender. Total paralysis is rare Cranial nerve disturbances are sometimes seen; occasionally bladder involvement will be indicated by retention and overflow leakage The spinal fluid protein is elevated sometimes to as high as 300 mg, but the cell count is normal. The course is progressive unless the diabetes can be brought and maintained under proper control, whereupon gradual disappearance of symptoms and signs is to be expected within a matter of months. In a certain number of diabetics who are asymptomatic, careful examination will show, uni- or bilaterally, minor objective sensory changes in the foot and loss of ankle jerk which are thought to represent a mild form of neuropathy. This picture may exist for years without progression.

Porphyric Neuropathy

The appearance of polyneuropathy in the absence of obvious cause should always suggest the possibility of porphyria. This is particularly true if there is



Fig 37 3 Diphtheric neuropathy with involvement of fifth, sixth, seventh, tenth and twelfth cranial nerves Patient is trying to smile and look toward left

Cutaneous Diphtheria. Especially common in the tropics, this often shows, days to weeks after onset, paralysis of the muscles supplied by the nerve root which also serves the area of the lesion As in the respiratory form, generalized polyneuropathy indistinguishable clinically and by laboratory studies from acute idiopathic polyneuropathy may develop, usually within 1–2 months Cases in this category are common among battlefield casualties.

Chemical Poisoning

Among the common chemicals causing neuropathy are lead, arsenic, inorganic phosphorus, methyl alcohol, trichlorethylene, thallium, carbon tetrachloride, trinitrotoluene, aniline and its derivatives, and carbon monoxide. Immune serum and certain of the newer therapeutic agents are responsible for some cases In recent years organic phosphates, used in many insect sprays, have become a hazard in the home as well as elsewhere When peripheral neuropathy is evident or suspected, careful investigation to exclude occupational or home exposure to any of these noxious substances is imperative Depending on the offending agent, the neuropathy may be predominantly motor, sensory, or mixed In adults, metallic lead and the oxides usually affect the extremities; volatile lead causes encephalopathy. In children, all types produce the latter. When the extremities are involved, weakness of the arms alone or of arms and legs is the first complaint; deep reflexes are absent and wrist drop or foot drop is common. Pain is mild or absent. Other characteristic features are lead line on the gingivae (*see* Chap. 4), bouts of intestinal colic, and characteristic changes

in the blood, the most important of which is stippling. Lead encephalopathy is characterized by headache, loss of memory, aphasia, pareses, convulsions, and in acute cases, by profound delirium, mania, or coma.

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Porphyric Neuropathy

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a history of paroxysms of acute abdominal pain occurring independently or in association with neurologic symptoms, or of passage of burgundy-red urine. The neurologic changes vary from mild paresthesia and weakness of one or more extremities to complete paralysis. Sensory loss is rare Cranial nerve involvement may be indicated by dysarthria, dysphagia, or facial paralysis. Psychiatric symptoms sometimes develop during an attack Death from respiratory paralysis can occur. The cerebrospinal fluid is normal Episodes may be precipitated or aggravated by administration of barbiturates, sulfonamids and certain other drugs The diagnosis can be confirmed by demonstration of porphyrins in the urine

Polyarteritis Nodosa

Multiple peripheral neuropathy is frequently seen in polyarteritis nodosa, less often in disseminated lupus erythematosus Especially during the later stages one finds evidence of sensory, motor, or mixed impairment in one or more extremities Sometimes the trunk is involved There is a characteristic tendency for improvement to occur in one region while further trouble develops in another Spinal fluid changes are minimal to absent

Leprosy

An early diagnostic criterion is anesthesia, at first thermal and light tactile, appearing in one or more areas It may precede appearance of any objective cutaneous change Another is cord-like thickening or nodularity of a nerve trunk, most likely the great auricular, ulnar, median or any one of a leg Nonspecific neuralgic pains may be experienced before the sensory loss As the disease progresses, anesthesia in the affected areas becomes complete, and weakness, paralysis, atrophy, trophic changes, and contractures of one or more extremities

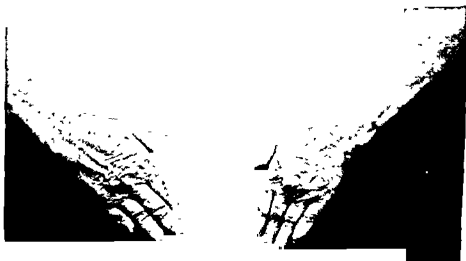


Fig 37 4 Leprosy. Deformities of fingers and loss of terminal phalanges due to neuropathy (Courtesy Dr. Thomas B Weller, Head, Dept of Tropical Public Health, Harvard University)

develop. The face is almost always involved. The soft tissues and bones of the digits are gradually absorbed, leaving the patient with stumps instead of fingers or toes. Ulceration is common as a result of a trophic disturbance or infection imposed on unnoticed burn or injury of an anesthetic part. Keratitis and subsequent ophthalmitis secondary to injury of the anesthetic cornea are common. The diagnosis of leprosy is confirmed by demonstration of the bacillus in smears or biopsy preparations from a cutaneous lesion. Nasal smears are no longer regarded as dependable.

Among the other disturbances of which polyneuropathy may be a complication are infectious mononucleosis, mumps, carcinoma of the lung, sarcoidosis, and amyloidosis. Rarely is it encountered—usually in a localized or limited form—following administration of an antiserum or, less often, a vaccine.

THE CRANIAL NERVES

Optic Neuritis

This is observed in various degenerative, inflammatory, and toxic disturbances, including hypertension, multiple sclerosis, encephalitis, neurosyphilis, and poisoning by carbon monoxide, lead, methyl alcohol, pentavalent arsenicals, and other chemicals. Sometimes no explanation is found. Loss of vision may develop rapidly; *in the severe case it can become complete within a few days.* The patient often complains of tenderness of the eyeball and pain, especially on movement of the eye. If the nerve lesion is close to the globe, one will find congestion of the disc, blurring of its margin, and other changes similar to those found in early papilledema (*see* Chap. 4). When the nerve is damaged more proximally, the disc may be normal (*retrobulbar neuritis*). Whether visual impairment is transient or permanent depends on the cause; in the progressive case eventual optic atrophy is likely.

Retrobulbar neuritis is often an early and sometimes the first manifestation of multiple sclerosis. A patient may experience one or more transient attacks over a period of several years before any other indication of multiple sclerosis appears.

Optic Atrophy

The acquired type has already been discussed (*see* Chap. 4). *Hereditary* primary optic atrophy (*Leber's disease*), occurring usually in males between 20 and 30, is almost always bilateral and slowly progressive. Occasionally it remains stationary. The disc is grayish-white, with sharply outlined margins and some cupping.

Trigeminal Neuralgia

Also known as *tic douloureux*, this is a unilateral disturbance of unknown cause occurring chiefly in older persons, especially women. It is marked by recurrent paroxysms of sudden, agonizing, lightning-like pain lasting a few seconds to minutes and accompanied by facial spasm and often by an outcry. Attacks vary

in frequency from many times a day to only one or two a month There may be long periods of complete remission. As a rule the pain is initially confined to the middle, lower, or upper part of the face, depending on which branch is affected; with time, it is apt to spread to the distribution areas of one or both of the other branches Especially characteristic is precipitation of an attack by some normally inconsequential stimulus such as the lightest touch to some part of the distribution area (*trigger point*), or some facial movement such as talking, chewing, or laughing To avoid initiating a spasm the patient learns to evade the trigger mechanism and thus may speak with his face immobile, refuse to eat or even to touch his face on the homolateral side There is no impairment of objective sensation or motor function.

A comparable picture often causing confusion can be created by an infected or impacted tooth, a retained root fragment, malarticulation of the temporomandibular joint (*Costen's syndrome*), an infected sinus, or histamine cephalgia In these disorders pain is neither so paroxysmal nor so obviously related to one or more trigger mechanisms

Neuralgia of the same character can also result from irritation of the sphenopalatine or glossopharyngeal ganglion The former causes pain about the eye referred posteriorly toward the postauricular region and extending downward into the neck; the latter, pain often precipitated by swallowing, in the pharynx, base of the tongue, and sometimes the ear

Bell's Palsy

This term refers to peripheral facial nerve paralysis of unknown cause It is thought to be due to edema of the nerve with resultant pressure due to swelling where it passes through the Fallopian canal. Exposure to cold or a draft is often blamed but it may well be that this is an effect, not a cause; the disease may make the patient aware of a draft which ordinarily would not be noticed Asymmetry of the face, impaired movement of one side, or irritation of or inability to close one eye may appear first, the picture of peripheral seventh nerve paralysis develops quite rapidly (*see* Chap 36). Recovery within 2 weeks to 6 months is the rule but it is often incomplete.

Vestibular Dysfunction

Impairment of labyrinthine function can be created by a disturbance of the labyrinth, the vestibular portion of the eighth nerve, or their central connections For convenience, labyrinthitis is included here because its manifestations are often so similar to those caused by lesions involving the nerve pathways

Acute Labyrinthitis. Strictly speaking, acute labyrinthitis refers to a disturbance of labyrinthine function caused by bacterial infection secondary to acute or chronic otitis media. However, the term as generally used also includes a group of cases of unknown etiology which present similar clinical findings; virus infection is thought to be responsible for many of these.

Onset is sudden. While walking, standing, sitting or rising from bed, the patient will be abruptly seized by intense vertigo, fall to the floor or bed, become

nauseated, perhaps vomit, and break into a cold sweat. He will experience a feeling of rotating within his environment or of his environment rotating around him, the sensation is so real that, if in bed, he will hold on to its sides for fear of falling out. Even if on the floor, he will have the urge to "hold on". Symptoms are less pronounced when he lies still with his eyes shut but promptly increase if he moves at all or opens his eyes. If he tries to walk, he will reel and promptly fall. Tinnitus, a feeling of fulness or pressure in the ear, and impairment of hearing are often present. Nystagmus, usually toward the contralateral side, and homolateral past-pointing will be found. In contrast to cerebellar ataxia, muscular incoordination is not a feature, the ataxia and related symptoms are a reflection of disturbed equilibrium. An episode will last from a few minutes to several days.

Motion sickness is also marked by nausea, vertigo and the other indications of labyrinthine irritation but the auditory components are lacking.

In a medullary vascular lesion or tumor or multiple sclerosis, symptoms and signs similar to those of acute labyrinthitis may develop but the course is longer and one will usually find other indications of a brain stem disorder.

Toxic Labyrinthitis. This occurs in acute alcoholism and from toxic doses of certain drugs, the most common being the salicylates, quinine, quinidine, and streptomycin. A few cases have been attributed to corticosteroid therapy. The clinical picture is similar to that described above except that onset, as a rule, is more gradual, the severity and length of the attack are influenced by the strength and duration of treatment.

Ménière's Syndrome. This term is applied to cases characterized by *repeated sporadic attacks* of labyrinthine dysfunction as described above. They are supposedly due to hydrops of the endolymphatic duct. They occur suddenly without any known precipitating factor, varying in duration from hours to days and in frequency from days to years. Persistent tinnitus and slowly progressive deafness are the rule. The former becomes more high pitched and the latter more pronounced during an attack, each tends to return to its former status once the episode subsides. Except for these two symptoms, the patient feels well between times. Caloric and turning studies usually show reduced vestibular function on the homolateral side. Audiometric tests will detect slight hearing loss in at least some frequencies before overt deafness becomes evident. These special studies should not be attempted during attacks because the patient's incapacity and distress make cooperation impossible.

Acoustic Neuroma. Acoustic neuroma or other cerebellopontine angle tumor is usually benign and slowly growing. As a rule, progressive deafness, often accompanied by tinnitus, precedes the appearance of other manifestations by months to years. In time, one will find further indications of pressure on the eighth nerve and surrounding structures—headache, vertigo, nystagmus, cerebellar ataxia and incoordination, and signs reflecting impairment of other homolateral cranial nerves, especially the fifth, sixth, and seventh. Diminution or absence of corneal reflex is frequently the first sign of additional cranial nerve involvement. Early, often pronounced, elevation of spinal fluid protein is the

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Acoustic Neuroma. Acoustic neuroma or other cerebellopontine angle tumor is usually benign and slowly growing. As a rule, progressive deafness, often accompanied by tinnitus, precedes the appearance of other manifestations by months to years. In time, one will find further indications of pressure on the eighth nerve and surrounding structures—headache, vertigo, nystagmus, cerebellar ataxia and incoordination, and signs reflecting impairment of other homolateral cranial nerves, especially the fifth, sixth, and seventh. *Diminution or absence of corneal reflex* is frequently the first sign of additional cranial nerve involvement. Early, often pronounced, elevation of spinal fluid protein is the

in frequency from many times a day to only one or two a month. There may be long periods of complete remission. As a rule the pain is initially confined to the middle, lower, or upper part of the face, depending on which branch is affected; with time, it is apt to spread to the distribution areas of one or both of the other branches. Especially characteristic is precipitation of an attack by some normally inconsequential stimulus such as the lightest touch to some part of the distribution area (*trigger point*), or some facial movement such as talking, chewing, or laughing. To avoid initiating a spasm the patient learns to evade the trigger mechanism and thus may speak with his face immobile, refuse to eat or even to touch his face on the homolateral side. There is no impairment of objective sensation or motor function.

A comparable picture often causing confusion can be created by an infected or impacted tooth, a retained root fragment, malarticulation of the temporomandibular joint (*Costen's syndrome*), an infected sinus, or histamine cephalgia. In these disorders pain is neither so paroxysmal nor so obviously related to one or more trigger mechanisms.

Neuralgia of the same character can also result from irritation of the sphenopalatine or glossopharyngeal ganglion. The former causes pain about the eye referred posteriorly toward the postauricular region and extending downward into the neck; the latter, pain often precipitated by swallowing, in the pharynx, base of the tongue, and sometimes the ear.

Bell's Palsy

This term refers to peripheral facial nerve paralysis of unknown cause. It is thought to be due to edema of the nerve with resultant pressure due to swelling where it passes through the Fallopian canal. Exposure to cold or a draft is often blamed but it may well be that this is an effect, not a cause, the disease may make the patient aware of a draft which ordinarily would not be noticed. Asymmetry of the face, impaired movement of one side, or irritation of or inability to close one eye may appear first; the picture of peripheral seventh nerve paralysis develops quite rapidly (*see* Chap. 36). Recovery within 2 weeks to 6 months is the rule but it is often incomplete.

Vestibular Dysfunction

Impairment of labyrinthine function can be created by a disturbance of the labyrinth, the vestibular portion of the eighth nerve, or their central connections. For convenience, labyrinthitis is included here because its manifestations are often so similar to those caused by lesions involving the nerve pathways.

Acute Labyrinthitis. Strictly speaking, acute labyrinthitis refers to a disturbance of labyrinthine function caused by bacterial infection secondary to acute or chronic otitis media. However, the term as generally used also includes a group of cases of unknown etiology which present similar clinical findings, virus infection is thought to be responsible for many of these.

Onset is sudden. While walking, standing, sitting or rising from bed, the patient will be abruptly seized by intense vertigo, fall to the floor or bed, become

nauseated, perhaps vomit, and break into a cold sweat He will experience a feeling of rotating within his environment or of his environment rotating around him; the sensation is so real that, if in bed, he will hold on to its sides for fear of falling out Even if on the floor, he will have the urge to "hold on". Symptoms are less pronounced when he lies still with his eyes shut but promptly increase if he moves at all or opens his eyes If he tries to walk, he will reel and promptly fall Tinnitus, a feeling of fulness or pressure in the ear, and impairment of hearing are often present. Nystagmus, usually toward the contralateral side, and homolateral past pointing will be found. In contrast to cerebellar ataxia, muscular incoordination is not a feature; the ataxia and related symptoms are a reflection of disturbed equilibrium. An episode will last from a few minutes to several days

Motion sickness is also marked by nausea, vertigo and the other indications of labyrinthine irritation but the auditory components are lacking.

In a medullary vascular lesion or tumor or multiple sclerosis, symptoms and signs similar to those of acute labyrinthitis may develop but the course is longer and one will usually find other indications of a brain stem disorder

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Onset is sudden. While walking, standing, sitting or rising from bed, the patient will be abruptly seized by intense vertigo, fall to the floor or bed, become

fected. *The initial paralysis may be all that develops* but involvement of other groups remains a hazard for a week or more. Although muscular pain, especially of the affected regions, is striking during the acute phase, objective sensory *changes do not occur*. Fever usually subsides with or shortly after onset of paralysis; its prolongation is an ominous sign in terms of extent and degree of paralysis. In the affected parts tendon reflexes, although sometimes increased at the start, *soon become diminished to absent* Paralysis of the diaphragm or intercostal muscles, most apt to occur when one or both upper extremities are involved, causes respiratory impairment, the patient will show rapid, shallow, labored breathing, with increasing utilization of his accessory muscles as vital capacity falls toward the critical level When breathing is seriously hampered, confusion with bulbar disease is likely (*see below*). Severe involvement of the lower extremities may be accompanied by paralysis of the bladder mechanism, which can disappear or become permanent In the latter event, infection and calculus disease of the urinary tract become an added hazard Fecal incontinence is common during the acute stage

In all but the most severe cases appreciable improvement can be expected if proper treament is promptly initiated and assiduously maintained Many patients will show complete recovery, or mild to moderate functional impairment of one or more muscle groups The ill-fated will end up with pronounced paralysis, muscle atrophy, absence of deep reflexes, and perhaps deformities due to contracture of muscles opposing those which are paralyzed, when the disease starts in childhood, impaired growth of the involved limbs adds to the handicap Continuous or intermittent dependence on respiratory aids is the tragic outcome of some cases of respiratory paralysis

Bulbar Type. In 10–15 per cent of cases or more, disease of the medulla occurs along with spinal involvement, or independently This form is more common in the older age groups At the start, restlessness and anxiety are striking. Paralysis of the palate and muscles of deglutition is indicated by nasal voice, regurgitation of fluids through the nose, dysphagia, and choking, ocular motor palsy or facial paralysis may result from involvement of the pertinent nerve *Breathing becomes jerky, irregular, and variable in depth and rate* In the pure bulbar form, the respiratory distress may be a reflection of medullary disease, the respiratory muscles themselves are not necessarily directly affected Death *from failure of the respiratory center, or secondary pulmonary infection* due to inadequate cough and consequent retention of secretions is likely If the patient survives, complete recovery without evidence of residual bulbar disease is the rule

In the active stage of poliomyelitis the spinal fluid is under normal or slightly increased pressure, sometimes if it is permitted to stand, a *delicate fibrin clot* will develop Its protein is normal or slightly elevated at the start but tends to increase further during the first 2–3 weeks. The cell count, highest in the preparalytic stage and rapidly falling after onset of paralysis, averages 50–300 per cu mm , polymorphonuclear leukocytes predominate during the first day or

rule. Labyrinthine function tests show diminished response on the affected side In the advanced case choked disc and other manifestations of elevated intracranial pressure are likely. When acoustic neuroma is suspected, support for the diagnosis may be obtained by x-ray, which often shows enlargement of the internal auditory meatus.

In many of the above disturbances, the process may proceed to complete loss of function. Here, vertigo, nystagmus and other irritative signs will disappear but deafness and tinnitus will persist and total absence of vestibular response will be demonstrable by the labyrinthine studies

THE SPINAL CORD

Anterior Poliomyelitis

Predominating in children and adolescents, this is an acute infectious viral disease It occurs sporadically or endemically throughout the year but reaches epidemic proportions in different regions from summer to summer Although the past quarter century or more has seen it become relatively less frequent in infants and decidedly more so in adults, especially those in the third decade, the highest incidence remains in childhood Pregnant women are especially susceptible Several strains of the responsible virus have been identified Infection by one confers immunity for that strain but not for the others Although statistics are incomplete, it would appear that the recent widespread use of polyvalent antipoliomyelitis vaccine has appreciably reduced the morbidity of the disease and perhaps its severity.

Non-Paralytic Type. The disease is ushered in by the symptoms of an acute infection—fever, malaise, mild headache, coryza, and perhaps nausea, vomiting, or diarrhea As a rule after 2–3 days increasing irritability, more severe headache, and muscle aches, especially in the neck and back, appear; stiffness of the neck and perhaps Brudzinski's neck or Kernig's sign will become demonstrable An appreciable number of patients appear to recover from the prodromal phase within 2–3 days, at this point they are likely to be regarded as having experienced only a mild respiratory or gastro-intestinal infection But 2–3 days later they again become ill with fever and the portentous symptoms and signs just described It is well established that in many instances complete recovery occurs within a week or so, paralysis does not develop Especially during an epidemic, any person presenting the above picture must be suspected of having poliomyelitis, unless paralysis develops, diagnosis can be proved only by demonstration of the spinal fluid changes indicated below

Paralytic Type. Early symptoms and signs are as described above. Flaccid paralysis of one or more muscle groups appears usually within 2–5 days after onset of the meningeal signs Occasionally it is delayed for a week or more, rarely, it is actually the first sign of trouble. In any particular muscle, paralysis reaches its maximum in 1–2 days One or both lower or upper extremities, the back muscles, or those of the shoulder or pelvic girdle are most commonly af-

Amyotrophic Lateral Sclerosis. In some patients beyond the age of 50, progressive muscular atrophy is complicated by degeneration of the corticospinal tracts In addition to atrophy, weakness, and fasiculation of the upper extremities, one finds spasticity, partial paralysis, increased deep reflexes, and Babinski sign in the lower extremities. Death within [illegible] years is the rule.

Subacute Degeneration of Spinal Cord

Also known as *combined system disease*, this occurs as a complication of pernicious anemia. It usually starts insidiously, rarely abruptly, either before or after the appearance of noteworthy change in the blood or any clinical stigma of pernicious anemia. Achlorhydria is always found. The underlying process is degeneration of the dorsal and lateral columns, sometimes of other tracts, the peripheral nerves, and, rarely, the cerebrum. The neurologic picture depends on the relative degree to which the various tracts are damaged.

The first symptom is apt to be numbness, tingling, or other paresthesia in the feet or hands; actual pain may occur, especially if there is peripheral nerve involvement. Loss of position sense is an important early symptom, in the hands it is indicated by clumsiness, in the feet, by ataxia which may be initially apparent to the victim only when he walks in the dark Sometimes weakness or stiffness of the legs appears first Later the patient becomes definitely unsteady and walks on a wide base with eyes pinned to the ground. As a rule his legs are spastic, with exaggerated reflexes and extensor plantar responses. If posterior column damage predominates, the legs show flaccidity and diminished to absent reflexes; plantar responses are usually extensor, rarely the opposite. Comparable changes in the upper extremities are not common although coordination of finer movements is almost always impaired.

Vibration sense is diminished to absent in the lower extremities, sometimes the upper Tactile, thermal and pain sensation are rarely affected. Mental changes are occasionally encountered—irritability, lack of cooperation, confusion, defective memory. Rarely one will find punctate hemorrhages in the retina, or optic atrophy

Although blood changes and other manifestations of pernicious anemia are expected, their absence, except for achlorhydria, does not exclude the disease. In recent years the widespread use, for non-specific "tonic" purposes, of preparations which include small amounts of vitamin B_{12} and folic acid has increased the chance of encountering a case with neurologic changes but without anemia.

If the disease is not recognized and properly treated, progression of neurologic and other changes will lead to total incapacity, urinary and fecal incontinence, and eventually death from inanition or superimposed infection Recovery can be effected and maintained by adequate therapy but, when especially severe, the neurologic manifestations may only partially disappear.

Tabes Dorsalis

See page 869.

lymphocytes later. In contrast to tuberculosis and other forms of bacterial meningitis, its sugar content is normal. *A normal spinal fluid, particularly a normal cell count, is almost never found in the pre- or early paralytic stage.*

Because of the danger of impairing resistance of the regional nerve cells, an elective operation, especially tonsillectomy or other operation in the nose or mouth, should not be performed during an epidemic, or even during the so-called "polio season". Furthermore, many authorities believe that immunization and other elective procedures requiring subcutaneous or intramuscular injection should be postponed whenever possible.

In recent years, a disease resembling poliomyelitis, *especially in its early* stage, has been encountered in this country and elsewhere (*benign myalgic encephalomyelitis*) Appearing chiefly in late summer and early fall, it begins with mild fever, headache, stiff neck, and generalized muscular aches, cramps, and tenderness. The muscle symptoms are more severe and last longer than in poliomyelitis Other prominent features are impaired memory, emotional lability, anxiety, sleeplessness, indications of brain stem involvement such as diplopia, nystagmus, deafness, or ocular motor palsies, and scattered areas of anesthesia Lateral tract involvement may be reflected by increased deep reflexes and extensor plantar responses Spotty muscular weakness is likely, atrophy is rare Enlargement of lymph nodes, liver, or spleen may be found *Spinal fluid is* normal Most patients recover completely within 2–10 weeks but some experience relapses intermittently for months to years Fatalities are rare

Progressive Motor System Disease

Progressive Muscular Atrophy. This disease, progressive bulbar palsy, and amyotrophic lateral sclerosis are probably variations of the same process, which is one chiefly affecting the motor nerve cells Preponderant in males, particularly *of the fourth and fifth decades, progressive muscular atrophy is due to degenera*tion of anterior horn cells. Initially it is usually manifested by weakness and atrophy of the muscles of one or both hands with subsequent involvement of the arms and shoulder girdle As a rule, one side *is affected earlier and more* severely than the other Fine, rapid, fasicular quiverings of the affected muscles are characteristic Deep reflexes are diminished or absent Ultimately, bulbar paralysis and sometimes involvement of the lower extremities develop The course is chronic and progressive with occasional partial remissions The patient eventually becomes bedridden and dies of respiratory failure or some intercurrent disease

Progressive Bulbar Palsy. Sometimes, especially in the older age group, the process starts in the medulla One finds progressive difficulty in speaking, swallowing, and chewing The tongue and perhaps the lower part of the face become atrophied and show fasiculations As a rule the patient dies of respiratory failure within *2–3 years.* If a case is of longer duration, involvement of the spinal cord may be superimposed, giving a picture similar to progressive muscular atrophy, or signs reflecting corticospinal tract impairment.

reflexes of the legs depends on the status of their arcs and the corticospinal tracts.

A lesion in the *medulla* creates paresis of the palate, uvula, tongue, pharyngeal muscles, and vocal cords. Other possible features are atrophy and fasciculation of the tongue, nystagmus, and impaired pain and temperature sensation of parts of the face.

Spinal fluid changes are not striking; protein content and cell count may show some elevation.

As a rule the course of syringomyelia is extremely prolonged. A stationary status may obtain for years; extension of the process can occur at any time. Minor remissions are possible The usual outcome is total incapacity with eventual death from intercurrent infection. In syringobulbia, death from involvement of vital centers is to be expected within months to a year or so.

Multiple Sclerosis

This disease is marked by multiple, patchy, scattered areas of demyelinization and gliosis which can occur in any part of the central nervous system. It is described with diseases of the spinal cord because lesions here usually lead to the most prominent and incapacitating manifestations. Although a progressive disturbance, it is characteristically featured by remissions and exacerbations of varying duration. Exposure to cold, any acute infection, trauma, and pregnancy have been accused of favoring or initiating exacerbations but whether the relationship is one of cause and effect or coincidence remains unestablished. Since areas of damage are so diffusely distributed and indications of trouble anywhere can disappear during remissions, no single clinical picture can be drawn. During the earlier stages symptoms are often so vague and ephemeral and signs so meager that the diagnosis of psychoneurosis is likely to be erroneously entertained for months or even years. Transient weakness, poor control, paresthesia or impaired sensation of one or more limbs, uni- or bilateral visual disturbance, diplopia, vertigo, and nystagmus are among the more common early manifestations Sometimes transient hemiplegia appears first Except in the relatively rare case where damage is severe from the start, any or all of these may clear partially or completely If the area responsible for any of these changes is involved in later exacerbations, less improvement is to be expected following each subsequent remission With time certain of them become permanent and, in addition, stiffness and definite ataxia develop Urinary frequency due to spasticity of the bladder, or urinary or fecal incontinence secondary to loss of sphincter control is likely Pain is not a feature.

In the typical case neurological examination will show one or more of the following variants strabismus or other evidence of oculomotor palsy, nystagmus, impaired visual acuity, optic atrophy, scanning speech, dysphagia, intention tremor of hands and feet, spastic paresis of one or more extremities, ataxia, absent abdominal reflexes, increased deep reflexes, ankle clonus, and uni- or bilateral Babinski or Hoffman signs. In a small percentage of cases predominance of posterior column involvement causes absence of reflexes in one or more extremities.

Syringomyelia

Cavity formation with associated gliosis begins in the region of the central canal of the cord and tends to spread horizontally as well as longitudinally. The disease usually starts at the cervical level, sometimes the lumbar, and is slowly progressive. As a rule the *well-established case will show a large lesion* in the cervical segment, the lumbar segment, or both, but sometimes there is *patchy, segmental distribution or involvement throughout the length of the cord.* A lesion in the medulla (*syringobulbia*) can occur independently or in association with a cervical lesion. The process usually encroaches first on the pain and temperature fibers crossing to the spinothalamic tracts. Later it extends in bizarre fashion to involve the anterior horn cells, lateral horn cells, corticospinal tracts, and perhaps the fibers of position and vibration sensation in the posterior columns. Although usually bilateral, it is not necessarily symmetrical.

Since cervical involvement usually predominates, symptoms are most likely to appear in the neck and upper extremities. Loss of pain and temperature sensation appears first; discovery of an unfelt burn of the finger or hand is often the first sign. Shortly after, destruction of anterior horn cells creates weakness, atrophy, and fascicular twitchings, first of the muscles of the hands, later of the arms and neck. If the lateral horns, which contain autonomic cells, become involved, one or more extremities will show vasomotor phenomena and trophic changes such as *thickening, atrophy or pigmentation of skin, atrophy or edema* of subcutaneous tissues, coarseness of nails, and indolent, painless ulcers of the fingers. One or more terminal phalanges or even entire digits can eventually be destroyed. Neurogenic arthropathy, especially in the shoulder and elbow, may develop (*see* Chap. 35). Deep reflexes are usually absent in the upper extremities. If a corticospinal tract is damaged, the lower extremity will show spasticity, increased reflexes and a Babinski sign unless the pertinent reflex arcs are interrupted by a coexistent lesion *in the lumbar cord.*

In disease of the *lumbar* cord, changes comparable to those described in the arms are found in the pelvic region and lower extremities. Paralysis of the bladder mechanism is likely. The status of the plantar responses and the deep



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Fig 37.5 Syringomyelia Patient is trying to raise both arms with hands open Note atrophy of shoulder girdle and left upper extremity.

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Diminution or loss of position and vibration sense in affected regions is the rule; patchy, ill-defined areas of impaired tactile and pain perception may be detected.

Emotional instability, personality changes and sometimes definite psychotic deviants such as memory loss, disorientation, confusion, and poor judgment are not uncommon in the well-established case Occasionally some of these will appear months or even years before other indications of the disease It is in the group showing emotional instability and indefinite areas of sensory loss prior to development of the more striking signs that the erroneous diagnosis of hysteria or psychoneurosis is likely to be made.

In multiple sclerosis the spinal fluid may be entirely normal or show one or more of the following variants. moderate elevation of protein, increase of lymphocyte count to as high as 100 per cu mm., slightly or definitely abnormal gold curve, characteristically first zone

The duration of the disease is extremely variable The usual pattern is one of gradual downward progression marked by remissions and exacerbations over a period of 10–20 years with ultimate total incapacity and death from inanition or infection An acute form with fatal termination within a few months to a year or so is occasionally encountered

Friedreich's Ataxia

The heredo-familial tendency is strong; several children in one family may be affected. The process is one of degeneration of fibers in the posterior and lateral columns of the cord. Since important cerebellar tracts are usually involved, cerebellar symptoms and signs are likely to predominate Manifestations, usually beginning during childhood, include ataxic gait, poor coordination, dysarthria, tremors of the arms and head, absent knee and ankle jerks, and extensor plantar reflexes Position and vibration senses are impaired Kyphoscoliosis and pes cavus, the causes of which are not clear, are usually found. Optic atrophy is a rare complication The course is chronic and slowly progressive. With time, loss of control reaches the point where movements are jerky and non-synchronized, the gait is reeling or lurching, and even gross activities of the arms and hands are uncoordinated The spinal fluid is normal Death results from incidental infection or involvement of the bulbar centers. Not all cases in the same family present such a serious picture; some show only one or two features of the disease.

A number of other hereditary syndromes, closely related to Friedreich's ataxia and marked by comparable degenerative changes in the central nervous system, have been described.

Segmental Lesions of Spinal Cord

The picture of transverse myelopathy is encountered in a variety of circumstances, among the most important of which are:

1. Partial or complete transection of the cord as by a penetrating missile or fracture or fracture-dislocation of a vertebra, traumatic or spontaneous.

2. Hemorrhage into the cord (*hematomyelia*). Usually following trauma, this may occur with or without fracture or dislocation of a vertebra.

3. Concussion of the cord. This occurs with a direct blow to the spine but without actual injury to the cord. Symptoms and signs are usually transient.

4. Compression by vertebral fracture or fracture-dislocation, tumor, epidural abscess, or any inflammatory lesion of the meninges leading to adhesions (*arachnoiditis*). The last-named may occur as a complication of meningitis or, rarely, injection into the subarachnoid space of serum, spinal anesthetic, or other agent. Infarction due to pressure on local surface or intrinsic vessels is often a feature of a compressive process.

5. Infarction secondary to impaired circulation in its nutrient vessels, as by thrombosis complicating meningitis, or compression by tumor or local abscess.

6. Multiple sclerosis, syringomyelia, subacute combined degenerative and other intrinsic diseases of the cord In any of these disorders, before the usual clinical features are well established, widespread involvement of the cord in a limited segment occasionally will create a picture indistinguishable from that of a local segmental lesion Rarely, transverse myelopathy occurs without discoverable cause

The type of onset and subsequent course depend on the cause The signs and symptoms reflect the location and extent of the lesion as much as its nature. The following facts may help to localize the trouble·

1 Segmental or radicular disturbances such as hyperesthesia, root pain, and localized tenderness occur at the approximate level of the lesion

2. Descending degeneration of the corticospinal tracts leads to motor impairment with typical spastic paralysis and increased reflexes below the lesion

3. Sensory impairment occurs below the level since the ascending (sensory) tracts are cut off from the higher centers

4 X-ray of the spinal canal following injection of a contrast medium may demonstrate block or a filling defect

The cerebrospinal fluid may be normal If, as often happens, the lesion has caused subarachnoid block, the spinal fluid pressure will be low and show slow rise on jugular compression and slow fall to normal after compression is released (When slow response to jugular compression and release is observed, one must exclude partial obstruction of the needle-tip by some agent such as a fold of arachnoid, before subarachnoid block can be regarded as *certain* If the needle is open, firm pressure on the abdominal wall will cause a prompt rise of pressure in the manometer, whereas if it is blocked, this change will not occur) With obstruction the protein content is usually elevated and often high enough to cause xanthochromic fluid and perhaps spontaneous clotting.

THE CEREBELLUM

Cerebellar disease may be congenital or acquired, acute or chronic Among the causes of *acquired disease* are trauma, encephalitis, other infectious processes, tumor, abscess, vascular accident, and perhaps unknown toxic agents. Signs of cerebellar dysfunction are also found in multiple sclerosis, *tumor of*

the cerebellopontine angle, Friedreich's ataxia, sometimes arteriosclerosis, and certain rare diseases

Attention is directed to the cerebellum and its tracts by cerebellar ataxia and muscular hypotonia (*see* Chap 36) In general the signs are the same whether the damage is to the structure itself or to the pathways leading to or from it If a lesion is in the cerebellum, the signs are homolateral; if in the tracts they are homo- or contralateral, depending on which ones are involved and at what level As a rule, the patient with cerebellar ataxia is not vertiginous In contrast, labyrinthine disease causes both vertigo and ataxia but the latter is a reflection of an impaired equilibrium mechanism, not of incoordination; even during an attack of vertigo, control of the limbs is not impaired If a labyrinthine disturbance is excluded, a combination of the two usually indicates disease involving both the cerebellum and its brain stem connections. Lesions in the anterior lobe are responsible for hypotonia, ataxia, and sometimes weakness and easy fatigability The hypotonia, more marked in the proximal muscle groups, may be obvious as hypermobility on passive movement, or be indicated by a pendular knee or triceps jerk In a lesion of the middle lobe or its pathways as they pass through the mid-brain, the patient may experience recurrent, brief, or prolonged tonic seizures in which the arms and legs are rigidly extended, usually with the arms strongly pronated and internally rotated (*cerebellar fit*) Serious disease, especially metastatic tumor or medulloblastoma, can exist in the cerebellum, yet the patient will show no ataxia or other sign of incoordination unless he stands up. Here the lesion is likely to be in the flocculonodular lobe

Developmental Malformations

Various types of malformation occur, sometimes independently, sometimes in association with anomalies elsewhere in the nervous system and/or defects of bone and overlying structures. Abnormal development of the cerebellum is one of the causes of hydrocephalus The most common is the *Arnold-Chiari* syndrome in which there is downward elongation of the brain stem and cerebellum into the cervical canal, hydrocephalus, and spina bifida occulta or meningocele, most likely in the lumbar region, are often associated Clinical manifestations usually appear early in infancy and reflect the hydrocephalus or damage to the cerebellum and brain stem Occasionally they develop in adult life, whereupon the picture can be confused with that caused by multiple sclerosis, syringomyelia, or posterior fossa tumor

Progressive Cerebellar Degeneration

Under this heading are included a number of syndromes, almost always heredofamilial, which are marked by symptoms and signs of progressive cerebellar degeneration with or without those of comparable changes in the brain stem and cord. Since overlapping is the rule, no clear-cut classification is possible although specific syndromes have, to some extent, been associated with onset at particular ages Only the most clearly defined are described.

Olivo-Ponto-Cerebellar Atrophy. Onset in late mid-life is the rule. One finds progressive ataxia of trunk and extremities, scanning speech, nystagmus, and other signs of cerebellar disease with, later, rigidity, mask-like facies, and tremor. The reflexes are normal. The course is slowly progressive, with incapacity developing after 5–10 years or more. Mental deterioration is a likely late complication.

Friedreich's Ataxia. (*See above.*)

Parenchymatous Cerebellar Degeneration. No hereditary factor is evident. The syndrome is marked by the appearance in mid-life of unsteadiness on walking followed by increasing ataxia and intention tremor of the legs, and eventually frequent falling. As a rule, the changes are confined to the lower extremities but in some cases the uppers may gradually become involved. Nystagmus is unlikely. The reflexes are normal. The course is slowly progressive but total incapacity does not necessarily develop.

Multiple Sclerosis

Although this disease has been described under spinal cord (*see above*), it is also listed here to emphasize that the cerebellum or its connections with the brain stem are involved in a high percentage of cases so that manifestations such as scanning speech, nystagmus, and ataxia of limbs are often clinically predominant.

Cerebellar Vascular Accident

Circumstances surrounding cerebellar hemorrhage, thrombosis, and embolism are comparable to those pertaining to the cerebrum (*see below*). Onset is sudden in hemorrhage or embolism, sudden, gradual, or by stages in thrombosis.

Hemorrhage. The signs reflect trouble in one or both hemispheres, such as nystagmus, intention tremor, incoordination, and ataxia. Extension of bleeding into the pons, a common complication, will be indicated by ocular palsies, dysphagia, paresis of one or more limbs, or other signs of impairment of cranial nuclei and/or the long tracts which traverse the pons and medulla. The spinal fluid is usually bloody due to extension of bleeding into the fourth ventricle or subarachnoid space. Hemorrhage into the cerebellum or brain stem has a high mortality rate.

Thrombosis or Embolism. Here, too, symptoms and signs referable to the cerebellum are apparent. Since the cerebellar arteries also supply the lateral portions of the brain stem, occlusion of a proximal segment of one of them will also create changes which reflect damage to this structure. When the *posterior inferior* artery is the site, onset is marked by severe vertigo with nausea and vomiting. Prominent features are nystagmus, paresthesia, and diminished pain and temperature sensation on the homolateral side of the face, contralateral extremities, and contralateral side of the trunk. Signs indicating involvement of the homolateral lower cranial nerve roots, especially vocal cord or palatal paralysis, and ataxia of the homolateral extremities are the rule. Homolateral

Horner's syndrome, reflecting damage to the descending sympathetic fibers in the medulla, may be observed. In *superior* cerebellar arterial obstruction, associated involvement of the lateral portion of the midbrain, if it occurs, will be indicated by impaired pain and temperature sense of the face, body, and extremities on the contralateral side, and choreiform movements of the homolateral arm and perhaps leg Obstruction of the *anterior inferior* artery will create homolateral deafness, facial paralysis, and sensory loss, homolateral Horner's syndrome, and impaired pain and temperature sense of the extremities and trunk contralaterally.

Cerebellar Abscess

This is most often due to direct extension from an infected middle ear or mastoid but is sometimes metastatic from a focus elsewhere, particularly a lung Systemic signs of infection vary from minimal to striking, depending on the acuteness of the underlying process In recent years antibiotics have tended to obscure the picture by partially suppressing the systemic manifestations or those related to the primary focus Occipital headache, sometimes with stiffness of the neck, is common Nystagmus and ipsilateral cerebellar signs are found Compression of the brain stem may create impairment of one or more cranial nerves. Spinal fluid is under increased pressure and usually shows elevation of protein and cell count.

Cerebellar Tumor

In childhood, primary tumor is more frequent in the cerebellum than in the cerebrum; in adult life, the reverse is true As a rule, cerebellar tumor in mid-life or beyond is metastatic

Medulloblastoma. Usually encountered in childhood or youth, this has a tendency to arise in midline posteriorly and extend into the hemispheres Motions of the arms and legs are normal when the patient is lying down but ataxia of limbs and trunk develops on standing or walking Nystagmus is usually present Signs of intracranial pressure may develop early as a result of internal hydrocephalus due to impaired outflow of spinal fluid from the fourth ventricle. Changes reflecting metastatic spread to the cerebral hemispheres or spinal cord are not unusual

Other Cerebellar Tumors. In either children or adults these are most likely to develop in a lateral lobe and thus create the typical ipsilateral cerebellar signs, especially hypotonia and incoordination, and nystagmus Occipital headache is apt to occur early; the head may be tipped backward and toward the side of the lesion Signs of pressure on the lower cranial nerve roots may be found. In any cerebellar tumor elevated pressure and protein content of the spinal fluid is the rule.

Acoustic Neuroma. This and other tumors of the cerebellopontine angle have already been described (*see above*).

THE BASAL GANGLIA

Although the precise function of the basal ganglia is not known, it is thought that the diseases listed below are related to disturbances predominant in these structures.

Parkinson's Syndrome (Paralysis Agitans)

This develops in late life or late mid life from unknown cause, and in earlier age groups as a sequela of epidemic encephalitis. It is thought to result occasionally from brain injury, carbon monoxide, manganese or lithium poisoning, and, rarely, from brain tumor. In recent years a comparable picture has been encountered following prolonged administration of reserpoid drugs. Cases arising in older persons have been attributed to arteriosclerosis but the relationship is not well established. In general, the terms Parkinsonian syndrome and paralysis agitans are used interchangeably but many observers prefer to confine the latter to the idiopathic cases. Except for a few variants noted below which appear to be peculiar to the postencephalitic cases, the various forms show the same general picture. As a rule, one can distinguish between them, if at all, only by history.

Onset is insidious. The first manifestations are usually uni- or bilateral increase in muscle tone, a sensation of stiffness, and slowing of voluntary movement in the affected parts. Rigidity may be brought out by having the patient flex a limb against resistance; the movement will proceed by a series of jerks instead of smoothly (*cogwheel phenomenon*). Tremor appears early, first in the fingers and hands, then the jaw, tongue, and other parts, sometimes it is the initial sign. Unlike that of cerebellar disease it is diminished on voluntary movement. In the fingers it takes the characteristic form known as *pill-rolling tremor*. Finer movements such as writing and buttoning become increasingly difficult. Countenance, speech, carriage, and gait undergo progressive and characteristic alterations. The face becomes expressionless, eventually mask-like, speech monotonous, sometimes stuttery, and all movements slowed. The trunk is bent forward, or, less often, to one side or backward.

In the advanced case the voice is weak, enunciation rapid and indistinct; only with effort can the victim speak slowly and clearly enough to be readily understood. Difficulty in chewing and swallowing, and excessive salivation with drooling are common. The gait is slow and shuffling with the eyes focused on the ground. In time the patient experiences trouble in starting to walk, but once underway, because of his tendency to fall forward due to his abnormal carriage, he soon breaks into an uncontrollable trot (*propulsion or festination*) which he has equal difficulty in stopping. While he is standing, anything which tends to throw him off balance, such as a slight jolt or reaching upward, may cause him to shuffle backward (*retropulsion*). Sensation and deep reflexes are not altered. Spinal fluid is normal. The course is long and relentless; ultimately the patient becomes bedridden and succumbs to malnutrition, aspiration pneumonia, or other intercurrent illness.

The postencephalitic case often shows certain features not ordinarily associated with the others. Disturbance of ocular movements is likely, especially episodes of blepharospasm or uncontrollable deviation of the eyes upward or to one side for seconds to minutes (*oculo-gyric crises*). When not evident, blepharospasm can sometimes be precipitated by lightly tapping the forehead between the eyebrows (*glabellar sign*). Pupillary irregularity, inequality, or impaired response to light or accommodation may be found. Personality changes, especially impulsive behavior, are common.

Hepatolenticular Degeneration

Also known as *Wilson's disease*, this is a heredofamilial disease in which muscular rigidity, tremor, and other signs consistent with basal ganglial degeneration occur along with portal cirrhosis of the liver. The pathogenesis is not understood but it is believed to be related to an inborn disturbance of copper metabolism. Signs appear during the second or third decade, sometimes later. The neurologic manifestations usually but not necessarily precede those of liver impairment.

The tremor resembles that of Parkinsonian syndrome or multiple sclerosis, or the movements are choreiform or athetoid; initially they can be uni- or bilateral but eventually both upper extremities are involved. In the early stage abnormal movement may be brought out by having the patient extend his arms in front of his body, after a short latent period, flapping motions of the hands or the entire extremities appear (*wing beating*).

Eventually one will find generalized rigidity as in the Parkinsonian syndrome, torticollis or other deformity due to local spasm, dysarthria or dysphagia due to spasticity of the pertinent muscles, and perhaps bizarre movements of lower extremities. Convulsions and mental changes sometimes occur. Sensation is unimpaired. The tendon reflexes are normal or increased. In a high percentage of cases, careful examination of the eyes will reveal a ring of brownish pigmentation *in the cornea adjacent to the limbus* (*Kayser-Fleischer ring*). The urine shows increased excretion of copper and amino acids.

Although sometimes fatal within a few months, the disease is more likely to run a chronic course with gradual decline over a period of 5–10 years or longer. Death results from liver failure or complications attributable to the neurologic changes.

Sydenham's Chorea

Most often seen in children and young adults, usually females, this is almost always found in association with manifestations of rheumatic infection, to which it is generally regarded as being etiologically related. The earliest signs are emotional instability indicated by irritability, laughing or crying on slight provocation, and indications of poor coordination, such as a tendency to drop objects or spill food. Uni- or bilateral choreic movements—irregular, involuntary, and purposeless—may appear anywhere, most commonly in the upper extremities and face. In the latter, repeated twitching, smirking, or grimacing is

observed. The choreic movements are exaggerated by emotional stress but can sometimes be temporarily stopped. Coordinated activities such as buttoning or sewing are clumsily performed. An extremity may become so weak and flaccid that it is practically inert. In the severe case, the patient incessantly thrashes about the bed. Fever, leukocytosis, and increased sedimentation rate are present in the acute stage. Although the course is prolonged, spontaneous recovery is the rule. In a child, differentiation between mild or early chorea and simple nervousness or habit tic may be possible only after extended observation.

Huntington's Chorea

Also known as *chronic degenerative hereditary chorea*, this is a heredofamilial disease involving both the cerebrum and basal ganglia. It is marked by choreic movements and, later, progressive mental deterioration. The former, usually appearing in the third or fourth decade, begin in the face and upper arms, but eventually involve the whole body. At the start they may be attributed to restlessness or nervousness, especially since at this time short voluntary movements are fairly well executed. Later facial grimacing becomes continuous, smacking of the lips and tongue striking, and articulation progressively impaired. Pronounced jerking movements of the extremities and swaying of the trunk develop. The gait is marked by jerky, dancing movements and sudden stops, starts, and turns. Sensation and reflexes are not impaired. Spinal fluid is normal.

Irritability or apathy and loss of attention and memory are the first indications of mental change. Rarely, these precede the abnormal movements by 1–2 years or more. Later one finds impulsive behavior and intellectual decline. The disease progresses steadily for 10–20 years but may remain stationary for intervals of months. Total physical and mental deterioration is the end result.

THE CEREBRUM

Cerebral Hemorrhage

Cerebral hemorrhage, thrombosis, and embolism are often grouped together under the term *cerebral vascular accident*. With justification subarachnoid hemorrhage can also be included in this group, although the lesion is actually in the subarachnoid space (*see below*). Due to rupture of a vessel, cerebral hemorrhage usually occurs beyond the age of 45 and is uncommon except in the presence of well-established hypertension; the pressure elevation may be only moderate in patients over 70. Although any part of the brain is vulnerable, the basal ganglia and adjacent internal capsule are most often involved. Among the rare causes of massive bleeding are rupture of a congenital or mycotic aneurism or of a vessel in primary or metastatic tumor, some blood dyscrasia, and excess dosage of an anticoagulant.

Onset is sudden with rapidly developing restless to deep coma. If there is time, the patient will complain of terrific headache and vomit immediately beforehand. The catastrophe may be initiated by physical effort or mental excitement, especially a rage. Respirations become deep, stertorous, and perhaps show

Cheyne-Stokes or other form of irregularity. Pulse is slow. Mild to moderate fever is the rule. Because of peripheral circulatory collapse the hypertension may no longer be evident. Within a few moments of onset, hemiplegia appears as drooping of one side of the face and flaccid paralysis of the extremities The plantar reflex is extensor on the affected side and, in the early stage, sometimes bilaterally. The spinal fluid is under increased pressure, if, as often happens, the hemorrhage extends into a ventricle, it will also be grossly bloody

Deepening coma, progressive rise of temperature, pulse, and respirations, and death within a few hours to days are the rule. Rarely, partial recovery ensues, but the patient is left with a residual hemiplegia which soon becomes spastic and, when the lesion is on the dominant side of the brain, some degree of aphasia.

Cerebral Thrombosis

When attributable to atherosclerosis, its predominant cause, cerebral thrombosis usually develops beyond the age of 45 but, especially in diabetics, sometimes earlier. Other possible but infrequent causes are: a vascular inflammatory process, as in meningovascular syphilis, tuberculous meningitis, polyarteritis, temporal arteritis and lupus erythematosus; pressure on a vessel by an intracranial tumor; increased viscosity of the blood, as in polycythemia Rarely, in children, thrombosis develops during the course of a systemic infection

Both the general and focal manifestations are dependent on the size and site of the infarction which, in turn, depend on what vessel is occluded and to what extent Larger infarcts create some degree of paralysis, impairment of consciousness, and sensory change. Smaller ones may occur without key signs Although a major episode often starts without premonitory symptoms, a careful history may bring out that during the preceding days to months the patient had experienced transient, usually mild, episodes of confusion, aphasia, giddiness, impaired or double vision, or numbness or weakness of a limb Depending on the vessel involved, an attack is marked by appearance during minutes to hours of progressive paralysis, and/or sensory loss of the face or one or more limbs, dysarthria, aphasia, hemianopsia, or other focal signs Confusion or loss of consciousness may occur at onset, or during the period in which the focal signs are progressing Convulsions, general or Jacksonian, may occur, headache is rarely prominent Fever and alteration of respiration are not the rule except when the lesion is extensive Paralysis, when present, is flaccid in the early stages; the pertinent deep reflexes are diminished to absent. If the patient survives, the former usually becomes spastic, the latter increased, and clonus is likely. When a lower extremity is involved, Babinski sign occurs early and persists if there is residual damage, in the initial phase it is often bilateral even though paresis is unilateral. The spinal fluid is usually normal but may show slight elevation of pressure and protein content

Except when the infarction is extensive and/or collateral circulation is inefficient because of associated disease in the pertinent vessels, improvement is to be expected beginning within hours to days However, during the first week or so increase of trouble from further thrombosis is a decided hazard. Improve-

ment may slowly continue for several months but the patient is usually left with some permanent damage; only when the lesion is small and collateral circulation good can complete recovery be expected The possibility of future attacks of thrombosis, any one of which may be incapacitating or fatal, remains a source of concern. Especially in the older age group there may be repeated, transient attacks of confusion, giddiness, aphasia, or weakness or numbness of a limb which reflect thrombotic episodes in small vessels; emotional lability, progressive loss of memory, and other signs of senility are likely. When the infarction is large and collateral circulation inadequate, the patient will remain paralyzed, in coma, or otherwise incapacitated, and eventually succumb to malnutrition, aspiration pneumonia, renal infection secondary to prolonged catheterization, or other complication.

The more common syndromes seen in obstructive disease of the arteries supplying the brain are briefly described in the succeeding paragraphs

Middle Cerebral Artery. This is the most frequent site One finds hemiplegia, hemihypesthesia, perhaps homonymous visual field defects, and, when the dominant hemisphere is involved, aphasia. The face and arm are likely to be more severely affected than the leg As indicated earlier, the paralysis is initially flaccid, later spastic The sensory impairment, which is cortical in origin, is indicated by paresthesia, astereognosis, and loss of two point discrimination

Anterior Cerebral Artery. The manifestations are quite similar to those just described except that paralysis and sensory loss are more striking in the lower extremity than in the arm or face

Internal Carotid Artery. One or more transient attacks of hemiparesis, hemihypesthesia, and perhaps homonymous hemianopsia affecting the fields on the contralateral side are likely to precede a major occlusion. Sometimes the patient will experience a few to many short-lived periods of partial or total monocular blindness in the eye on the side of the lesion, any such episode is capable of becoming permanent When the occlusion becomes well established, the picture is otherwise essentially similar to that of middle cerebral artery obstruction

Support for the diagnosis may be obtained by detecting diminished to absent pulsation of the common or internal artery (*see* Chap. 5).

Basilar Artery. A major episode is often preceded by repeated mild, transitory attacks Major occlusion creates bilateral paresis of the legs or, when the process is extensive, quadriplegia Confusion or loss of consciousness may or may not occur Dysphagia, dysarthria, impaired movements of the tongue, and evidence of trouble in the upper cranial nerve centers, such as ocular palsy, diplopia, and facial paralysis and paresthesia, are the rule. During and after the attack, emotional lability, indicated especially by laughing or crying without reasonable provocation, is often a feature. During the acute stage, loss of sphincter control is likely, it may persist for some time. The prognosis is poor However, striking improvement sometimes ensues, especially if adequate anticoagulant therapy is promptly initiated

Cerebral Embolism

The most frequent sources are a thrombus in the left side of the heart in longstanding auricular fibrillation, predominant mitral stenosis or recent myocardial infarction, and a vegetation in acute or subacute endocarditis. Less commonly a clot will break away from a thrombus situated on an arteriosclerotic plaque in the ascending aorta or a carotid artery, or from one which has formed in a pulmonary vein in bronchopulmonary infection or carcinoma, or after intrathoracic operation. Rarely, an embolus will reach the brain from a systemic vein by way of an intracardiac septal defect (*paradoxical embolus*).

As in cerebral thrombosis, the clinical picture is dependent on the site and size of the vessel occluded. Onset is always abrupt. There can be all gradations of trouble, from slight dazing to complete unconsciousness, transient small-field defects to hemianopsia, and transient sensory or motor impairment to complete hemianesthesia or hemiplegia. A convulsion may occur at onset. Following the initial insult, edema of surrounding parenchyma is apt to cause temporary increase of symptoms and signs, but these diminish as the swelling subsides. Or, relaxation of the artery following its contraction from spasm at the moment of onset may permit the embolus to shift distally, subsequent bleeding through the damaged segment of the infarcted vessel wall can then cause further damage to the brain. Another hazard is formation of a thrombus in the artery proximal to the embolus with resultant impairment of blood flow to a larger segment of parenchyma. The spinal fluid is usually normal but occasionally shows moderate elevation of protein and several hundred red cells per cu mm.

In bacterial endocarditis multiple small emboli are likely. Since these are infected, abscess formation, diffuse encephalitis, or meningitis is a possible complication. Another possible outcome is formation of a mycotic aneurism in an involved vessel, with eventual rupture. Embolism due to an infected lung carries the same risks.

Barring one of the complications described, functional recovery is usually excellent except when a major vessel is occluded, this pertains especially in the younger age group without generalized vascular disease. When the underlying disturbance is not correctable the danger of subsequent embolism remains. The patient with a large embolus and/or poor collateral vessels may end up with serious residuals, even total incapacity or permanent coma.

Fat Embolism. Following major trauma, usually but not necessarily with one or more fractures, globules of fat occasionally enter the systemic venous circulation, pass through the pulmonary circuit, and terminate in the brain. There may be no primary head injury. Autopsied cases show fat droplets in the smaller vessels of the brain and, especially in the white matter, petechiae or minute zones of softening in the parenchyma adjacent to them.

After a latent interval of hours to days during which the patient appears to be getting along satisfactorily, he will develop delirium or somnolence, seizures, tremors, spasms, or other focal neurologic signs. In contrast to head injury, the

cerebral manifestations are likely to wax and wane, recrudescences presumably being caused by new showers of emboli. Sometimes the diagnosis is supported by appearance of petechiae in the skin or fat globules in the urine. The patient [illegible] completely, or be left with some residual focal impairment. The [illegible] breathing, and [illegible] hap. 27). Rarely, [illegible] death will result within hours to days after surgical [illegible] of the injuries; postoperatively, recovery from the unconscious state induced by the general anesthetic fails to occur.

Particularly when they appear in mid-life or earlier, signs of infarction not readily attributable to arteriosclerosis or other obvious cause demand thorough investigation, especially to exclude brain tumor or abscess, syphilis, and subdural hematoma.

The table below (Fig. 37 6) shows the important distinguishing features between cerebral hemorrhage and large cerebral infarct due to thrombosis or embolism. Exceptions to the rule may be encountered.

Generalized Cerebral Arteriosclerosis

Occurring in elderly persons, this is marked by sclerotic changes with thrombosis of the arteries, arterioles, or both, resulting in small, moderate, or large areas of infarction. When a larger vessel is involved, the picture is that of cerebral thrombosis as described above. Irrespective of the occurrence of any such

	HEMORRHAGE	THROMBOSIS	EMBOLISM
Age	45–65	45 and above Syphilis 25–45	Any
Background	Hypertension	Hypertension Arteriosclerosis Syphilis	Subacute bacterial endocarditis Myocardial infarction Auricular fibrillation Mitral stenosis
Onset	Sudden	Sudden or progressive	Sudden
Headache	Intense	Slight or absent	Slight or absent
Vomiting	Often	None	None
Mental status	Deep coma	Normal, or slight confusion to deep coma	Normal, or slight confusion to deep coma
Stertorous breathing	Usually present	Present or absent	Present or absent
Paralysis	Hemiplegia	Slight paresis to hemiplegia	Slight paresis to hemiplegia
Blood pressure	High (200 plus)	Normal, high, or low	Normal, high, or low
Spinal fluid pressure	High	Slightly elevated or normal	Slightly elevated or normal
Blood in spinal fluid	Usually	Absent	Absent
Course	Usually death	Often recovery	Often recovery

Fig. 37 6. Differential points in cerebral hemorrhage, thrombosis, and embolism. (Prepared with assistance of Dr. Charles S. Kubik.)

recognizable episode one finds gradually increasing irritability, emotional instability, tinnitus, impaired memory, orientation and judgement, repetitious conversation, faintness or giddiness especially on change of position, and perhaps mild confusion. Memory loss is greater for recent than remote events. Transient aphasia or pareses may occur In the late stages orientation, memory, and judgment become virtually to totally lost Impulsive behavior and delusions of infidelity are possible features Indications of arteriosclerosis elsewhere, including the retinae, are found The course is gradual and relentless, but symptoms, especially those of mental impairment, fluctuate from day to day; a well-defined attack of cerebral thrombosis can occur at any time

Senile Dementia

Clinically and pathologically, it is difficult to draw a sharp line between senile dementia and cerebral arteriosclerosis The former is not marked by episodes unless arteriosclerosis is also present, as well it may be; episodes reflecting arterial thrombosis point to the latter. The early symptoms are exaggerations of the changes seen in many older persons, such as memory loss, emotional instability, and, often, childishness and quarrelsomeness Increasing dependence on offspring for attention and entertainment is striking The patient shows progressive loss of memory, retention and orientation At first, partial insight activates attempts to conceal memory gaps by fabrication or change of subject In time one finds a decided tendency to live in the past, restlessness even to the point of wandering away from home, marked hypochondriasis, agitated depression, and delusions or hallucinations Loss of judgement may lead to behavior aberrations Occasionally, sexual unrest is a problem; it may even lead to a sexual offense Unless death from intercurrent disease intervenes, progressive deterioration ultimately leads to dementia, helplessness, and need for custodial care

Presenile Dementia

Alzheimer's Disease. Attributable to diffuse cortical atrophy, this is marked by progressive mental deterioration resembling the senile form but beginning between 10 and 60 In addition to the mental changes described above, one finds gradually developing aphasia, apraxia, and agnosia Seizures of some type may occur periodically Neurologic examination is negative except for minor reflex or sensory changes in the terminal stage Spinal fluid is normal In some instances the course is relatively short, with complete dementia and helplessness appearing in 3–5 years; in others, serious trouble is postponed for 10–15 years Once a case is well established, pneumoencephalography shows diffuse cortical atrophy.

Pick's Disease. This is much more rare, and cannot be clinically distinguished from Alzheimer's disease However, since it is featured by cortical atrophy confined to the frontal and temporal lobes, differentiation can be accomplished by pneumoencephalography

General Paresis

See below.

Hypertensive Encephalopathy

This term refers to the acutely developing central nervous system manifestations often encountered in cases of hypertension when the blood pressure suddenly rises to a point decidedly above its usual level. Although seen in hypertension from any cause, it is most apt to occur in acute or chronic glomerulonephritis, eclampsia, and malignant hypertension. It has been variously attributed to vascular spasm, edema, and petechial hemorrhages in the brain; when vascular changes are pronounced, as in the older age group with arteriosclerosis, thrombotic phenomena of the smaller vessels may be partially responsible. Increasing headache, vomiting, partial to total impairment of vision, convulsive seizures, stertorous breathing, and confusion often progressing to coma, develop in association with the rapid rise in blood pressure. The retinae usually show papilledema and perhaps hemorrhages and exudates. Especially in malignant hypertension, or in the older age group, one may find hemiplegia, aphasia, or other focal sign indistinguishable from that attributable to cerebral thrombosis. The spinal fluid pressure and perhaps its protein content are elevated. Outcome is dependent on the nature of the underlying process; if the pressure can be lowered, recovery with or without residual changes is likely, but otherwise the attack will be fatal.

Toxic Encephalopathy

Poisoning by lead, mercury, arsenic, carbon monoxide, and other chemicals sometimes produces a severe form of encephalopathy with a clinical picture of headache, vertigo, restlessness, delirium, and even coma. Convulsive seizures are often prominent in this group. Symptoms and signs reflecting damage to any part of the brain are possible.

Encephalitis

Encephalitis Lethargica. Presumably due to a virus infection, this was first recognized following the influenza epidemic of 1917–19, when its incidence was high, and was seen sporadically and in lesser epidemics for a decade or more thereafter. The disease, usually ushered in by acute upper respiratory infection, was characterized chiefly by gradual appearance of restlessness, headache, slight febrile reaction, tinnitus or vertigo, impairment of extraocular motor or perhaps other cranial nerves, and drowsiness, wakefulness, or disturbance of sleep rhythm. The patient might continuously sleep or remain awake for days, or remain awake at night and sleep by day. Ocular motor manifestations varied from transient diplopia to complete ophthalmoplegia. Other features were lid ptosis, pupillary variants especially loss of accommodation to distance, facial paresis, generalized muscular weakness, respiratory disturbances such as eleva-

tion or diminution of rate, or irregular rhythm, and persistent hiccough, yawning, or gasping. Evidence of increased intracranial pressure, with papilledema, or signs of mild meningeal irritation were often found. Occasionally onset was sudden with severe headache, delirium or coma, and cranial nerve palsies. Some patients died in coma. Others recovered completely but the majority improved only temporarily and later developed one or more postencephalitic sequelae, most commonly Parkinsonian syndrome. In children, behavior problems, and, in adults, psychic symptoms such as mild depression, slowing of the mental processes, irritability, or symptoms of a hypochondriacal nature appeared. Spinal fluid in the acute stage showed normal to elevated pressure, increased protein, and moderately increased cell count with lymphocytes predominating; after a few weeks it returned to normal.

In the opinion of most observers, cases of encephalitis which have occurred during the past two decades are not, strictly speaking, encephalitis lethargica but belong in one of the other groups discussed below.

Neurotrophic Viral Encephalitis. Attributable to different specific strains of neurotrophic virus (Japanese, St. Louis, eastern and western equine, and others) this has been encountered in epidemics in this country as well as elsewhere, during the past two decades or more. Depending upon the etiologic agent and its severity, cases are marked by fever, headache, drowsiness, stupor or coma, tremor, perhaps seizures, vomiting, stiff neck, and cranial nerve palsies or other focal neurologic signs. Spinal fluid changes, also dependent on the type and severity, include slight to moderate elevation of pressure, protein, and cell count. In the nonfatal cases mild to serious sequelae, including mental deficiency, cranial nerve or other palsies, and convulsions, are not uncommon. In contrast to encephalitis lethargica, subsequent Parkinsonian syndrome is rare.

Postinfectious Encephalitis. This is seen as a complication of mumps, measles, chickenpox, and other viral diseases, and following smallpox or rabies vaccination. Although sometimes fatal or leaving serious sequelae, cases in this group are more likely to be mild and recover without complications.

Rabies

This is considered a form of encephalitis. It is usually acquired from a bite by an infected dog or less often some other infected domestic or wild animal such as a cat, rabbit, rat, or squirrel. Symptoms appear anywhere from two weeks to several months after inoculation, the more extensive the wound and the closer to the brain its site, the shorter the incubation period and the more severe the disease. The early symptoms are increasing irritability, malaise, headache, sleeplessness, and anxiety. Within a few days fever, restlessness, terror, stridor, and dysphagia develop, the last two reflect pharyngeal and laryngeal spasm. Violent episodes of agonizing, exhausting clonic spasms which occur without impaired consciousness and cause the patient extreme anguish are provoked by the slightest stimulus such as a footstep or the draft from an opening door. Until recently, death occurred within a week from a convulsive seizure

or respiratory or cardiac failure. Currently, thanks to the use of curariform drugs, respiratory aids and other supportive measures, there is some hope for recovery.

Tetanus

The first complaint, which may not occur for 2-3 weeks following the injury, is a feeling of tension in the jaws. Difficulty in opening the mouth and tonic spasms of the masseter muscles (*trismus* or *lockjaw*) soon follow. Spasm of the [illegible] dysphagia. Later, there are tonic spasms of the muscles of the back and neck, with opisthotonos, and finally generalized convulsions which may be set off by the slightest stimulus. Respiratory impairment secondary to laryngeal spasm and rigidity of the respiratory muscles eventually develops. Some degree of fever is the rule. The tendon reflexes are exaggerated. The spinal fluid is normal except for perhaps some elevation of pressure Formerly death, usually from asphyxia secondary to respiratory impairment, occurred in a high percentage of cases. In recent years the mortality rate has been appreciably reduced by intensive antiserum therapy, more thorough wound debridement, antibiotics, anticonvulsant drugs, and other supportive measures.

Cerebral Abscess

This is a localized suppurative process caused by extension of infection from a diseased middle ear, mastoid, sinus, or wound of the skull, or by metastasis from a distant focus, especially a lung abscess, bronchiectatic cavity, or bacterial endocarditic vegetation Symptoms usually appear gradually and at first reflect the systemic effects of infection. Formerly septic fever, chills, anorexia, and prostration were the rule, but in recent years they have often been obscured by antibiotic treatment of the underlying process When the abscess becomes walled-off, subnormal temperature may ensue. As intracranial pressure increases, one finds severe headache with vomiting, mental dullness or stupor, slowing of pulse and respirations, choked discs, and perhaps general or local convulsions The focal signs are similar to those of brain tumor. In middle ear or mastoid infection the first sign of temporal lobe abscess is likely to be aphasia or a homonymous visual field defect. Spinal fluid pressure is usually elevated, protein moderately increased, and sugar content normal. Depending on a number of factors, including the degree of encapsulation of the lesion and its nearness to the meningeal or ventricular surface, the cell count can vary from normal to decidedly elevated As in the case of brain tumor, lumbar puncture should always be performed with the greatest care.

Cerebral Tumor

This can be primary, or metastatic especially from carcinoma of a breast, lung, kidney, or a malignant melanoma at any site. Headache is often but not necessarily the earliest symptom. In fact, it must be emphasized that personal-

ity change, one or more general or focal seizures, a visual field defect, aphasia, weakness or numbness of a limb, or some other focal sign will appear long before headache becomes a problem. When present, headache is general or local, tends to occur intermittently and is often increased by cough, sneeze, or straining; it is not necessarily related to the site of the tumor nor can it, with any degree of certainty, be differentiated from pain in the head due to numerous other causes. Although nausea and vomiting may accompany a severe attack, other classic indications of increased intracranial pressure such as choked disc, persistent or projectile vomiting, slow pulse and respiration, and impaired consciousness are often late manifestations. One should hope to establish diagnosis before these signs develop. When a tumor is so situated in a cerebral hemisphere that hemiparesis is not an early sign, progressive contralateral weakness will eventually develop as the lesion spreads to involve the motor cortex; if the dominant hemisphere is the site, aphasia will also appear. Although details of tumor localization are without the scope of this book, distinctive signs pointing to various sites are briefly recorded. These may be misleading if there is high elevation of intracranial pressure or distortion of the brain by a large growth.

Frontal Lobe. Blunting of intellectual faculties, retardation of thinking, silliness and *joking out of keeping with the situation*, untidiness, or other indications of impaired judgment often develop first. As the tumor spreads posteriorly, involvement of the motor cortex creates progressive *contralateral hemiparesis* with aphasia if the dominant hemisphere is involved. Rarely, if the palmar surfaces of the patient's hand and fingers are stroked or an object such as a reflex hammer is placed in his hand he will exhibit a strong grasping motion (*grasp reflex*).

Parietal Lobe. Since this lobe contains the motor and sensory gyri, contralateral paresis and sensory deficits appear early. Jacksonian seizures are common; they may precede the motor and sensory impairment.

Temporal Lobe. The most characteristic sign of temporal lobe tumor is a generalized seizure preceded by an aura of an unpleasant odor (*uncinate seizure*). Aphasia is an early sign in disease of the dominant hemisphere. If and when the optic tracts become involved, partial to total contralateral homonymous hemianopsia is demonstrable.

Occipital Lobe. A progressive contralateral homonymous field defect is usually the first sign. General or focal seizures are common; they are often preceded by flashes of light or other visual aura. Visual aphasia usually develops.

Pituitary Body. Here one finds bitemporal hemianopsia, optic atrophy, and changes reflecting increased or diminished pituitary function such as giantism, acromegaly, Cushing's syndrome, diabetes insipidus, or panhypopituitarism.

When brain tumor is suspected, lumbar puncture must be performed with extreme care in order to lessen the chance of creating a "compression cone". Jugular compression tests must never be tried (see Chap. 36). The fluid is under increased pressure; moderate elevation of protein is likely. To confirm the diagnosis and localize the tumor it is often necessary to perform one or more of the specialized investigative procedures—electroencephalography, radioactive iso-



Fig 37 7 Metastatic carcinoma of brain Vertebral arteriogram showing abnormal concentration of tortuous arteries in tumor mass in occipital lobe (Arrow at top indicates area of bone destruction also due to a metastatic lesion)

tope concentration studies, arteriography, pneumoencephalography, ventriculography The last three are not without risk to the patient.

Post-Traumatic Syndrome

Trauma to the brain may be followed within days, weeks, or months by the appearance of such complaints as headache, dizziness especially on change of position, irritability, inability to work or concentrate, easy sweating, and emotional lability A previously healthy, reliable worker can become complaining and undependable; frequently the change is incorrectly ascribed to neurosis, malingering, or desire to collect insurance In other cases, memory loss, increased sensitivity to alcohol, sometimes chronic alcoholism, and serious behavior irregularities are encountered. Convulsive seizures may appear, if they start within a few days to weeks following the injury, it is presumed that latent idiopathic epilepsy has been brought to light. Depending on the location of the lesion, abnormal findings such as tremor, asymmetry of facial movements, unilateral exaggeration of deep reflexes with equivocal plantar response, and evidence of autonomic instability may be found. If a large lesion is present, a gross change such as psychosis, paresis, aphasia or hemianopsia may develop Whenever focal signs are found, subdural hematoma must be excluded.

Pellagra

Occurring as a result of inadequate diet, or poor absorption as in alcoholics, this is manifested first by headache, dizziness, nervousness, irritability, vague pains, digestive disturbances, and diarrhea. The tongue is painful and red. A characteristic eruption appears on the extremities or elsewhere (*see* Chap. 6). Sometimes there is evidence of damage to the peripheral nerves or spinal cord, especially its posterior and lateral tracts. In severe cases, agitation, depression, delirium, or stupor are likely.

Feeblemindedness

Due to defective cerebral development, this may be associated with congenital defects of the brain, cretinism, Mongolian idiocy, hydrocephalus, encephalitis periaxialis diffusa (Schilder's disease), early cerebral trauma, and other disorders. The degree of mental impairment is determined by intelligence tests In *idiocy*, mental development does not exceed the third year level; in *imbecility*, the seventh, in the *moron*, the twelfth The so-called adult level is 12 years. The clinical picture varies with the degree of impairment In general, these patients develop slowly in infancy; walking and talking come late, school history shows poor performance or failure Environmental maladjustment and behavior problems are common. In adult life these unfortunates are child-like and docile but easily angered and easily appeased Those with lesser deficits are usually able to perform simple routine work Idiots and imbeciles require custodial care The trend in recent years has been to avoid the terms indicated above, and to speak of severe, moderate, or mild mental deficiency.

THE MENINGES

Subarachnoid Hemorrhage

The classic case is due to a rupture of an aneurysm situated at a bifurcation of the circle of Willis or adjacently on one of its branches or tributaries As a rule the aneurysm is congenital, sometimes, syphilitic or mycotic Rupture usually occurs in young to mid-adulthood Hypertension is often but not necessarily in the background Among other causes of subarachnoid bleeding are traumatic rupture of a meningeal vessel, intracranial tumor, a blood dyscrasia, and extension from an intracerebral or subdural hemorrhage

Onset is abrupt with terrific headache which initially may be confined to the occipital region or homolateral side of the head but soon becomes generalized, the episode may or may not be induced by extra exertion or physical strain In some cases, headache of lesser severity precedes the major insult by hours to days. Stiff neck and other signs of meningeal irritation are the rule but they may not appear for the first day or so, later they may be masked by stupor The patient soon becomes confused to stuporous, deep coma, a bad prognostic sign, occurs only in the most severe cases Convulsions may occur. Papilledema and retinal hemorrhages are sometimes found The tendon reflexes can be depressed or hyperactive Moderate fever is present for the first few days If the hemorrhage is due, as it often is, to aneurysm of the internal carotid artery, resultant third

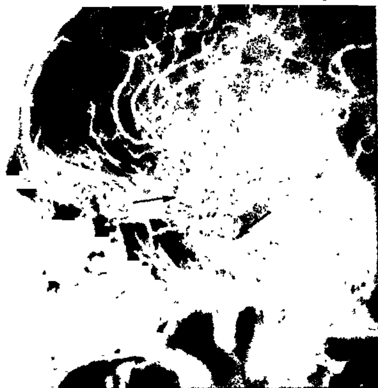


FIG 37 8. Aneurysm of anterior cerebral artery Carotid arteriogram Lesion indicated by upper arrow, internal carotid artery, by lower

nerve paralysis will cause diplopia, homolateral lid ptosis, inability to adduct the eye and dilatation of the pupil with failure to respond to light Monoplegia, hemiplegia, aphasia or other focal signs may develop as a result of bleeding into the brain parenchyma The spinal fluid shows elevated pressure (200–600 mm) and is uniformly bloody

The outcome is usually unpredictable Some patients die within hours to days, some survive the initial episode but die within weeks to months from a second rupture, others appear to recover completely but the hazard of a subsequent attack remains Often the aneurysm can be located by arteriography, this procedure is essential in cases which are regarded as perhaps favorable for surgical intervention.

Epidural Hematoma

Following head injury almost always with fracture of the temporal bone, hemorrhage may occur at the site of the blow The middle meningeal artery is the one most often injured After a period of unconsciousness lasting a few minutes to perhaps an hour (concussion) the patient appears to recover But a few hours later he develops delirium or drowsiness, convulsions, hemiplegia, and within 1 to perhaps 3 hours, stupor which rapidly progresses to coma. Slow,

stertorous respiration and slow pulse are the rule; papilledema may develop. Spinal fluid is under greatly elevated pressure and usually bloody, but occasionally clear. Prompt recognition of epidural hemorrhage is essential because, as a rule, the lesion is surgically correctable if operated upon in its early stage. Once the patient lapses into coma, the prognosis becomes rapidly less favorable even though operative intervention is undertaken.

Subdural Hematoma

This is another possible effect of head trauma. Especially in the older age group, the blow may have been mild. It must be emphasized that inability to obtain from the patient any history of injury does not exclude the diagnosis because the lesion itself often dulls his memory for past events including the traumatic incident. Sometimes onset is acute with impairment of memory, headache, dizziness, nausea, vomiting, unilateral seizures, hemiplegia, and perhaps stupor developing rapidly within hours to 1–2 days after the injury. Choked discs may be found. *In most cases the manifestations do not come to light until days to weeks after the injury*, here progression of the picture is slower than in acute hematoma, with headache and irritability existing for some time prior to gradual appearance of the more striking signs just described. Spinal fluid is under increased pressure; although sometimes clear, it is usually bloody in the early stages, later xanthochromic.

The diagnosis of cerebral thrombosis is often wrongly made, especially in those with arteriosclerosis and hypertension, because its clinical picture is so similar to that of subdural hematoma. The latter is overlooked because the blow on the head was either slight or not recalled by the victim. Elevated spinal fluid pressure favors a diagnosis of subdural hematoma, if it is more than 350 mm., cerebral thrombosis is excluded. Although the special procedures such as arteriography and air studies may be helpful, they are sometimes inconclusive, at times the serious condition of the patient makes them unwarranted. The diagnosis of subdural hematoma can be established with certainty only by uni- or bilateral burr hole exploration; if no lesion is found at the site of the injury, the opposite side of the head must be explored because a hematoma may be located there (*contrecoup*).

Acute Meningitis

This is most commonly due to meningococcus infection which appears sporadically or in epidemics. It can also be created by a local suppurative process such as otitis media, mastoiditis, sinusitis, brain abscess, or penetrating skull injury, or appear as a complication of an acute systemic infection such as pyogenic pneumonia, typhoid fever, and influenza.

Onset is acute with fever, perhaps chills, leukocytosis, headache, restlessness, irritability, and often photophobia, sensitiveness to noise, dizziness, or vomiting. The neck is stiff, Kernig and Brudzinski signs develop, and in severe cases the head becomes retracted. In contrast to musculoligamentous irritation the cervical stiffness is confined to anterior and posterior movement; other motions

are not appreciably hampered. Various reflex changes, paralyses, and ocular signs are found; convulsive seizures and coma are not uncommon. The spinal fluid is under increased pressure, cloudy due to presence of a large number of polymorphonuclear leukocytes, sometimes frankly purulent, and has a high protein, low sugar content. The offending organism can usually be detected by direct smear or culture.

Meningococcus infection usually shows a characteristic skin eruption (*see* Chap 3); sometimes this precedes meningeal signs by 1–2 days or more. Involvement of one or more joints is common In the fulminating case, signs of overwhelming systemic infection sometimes develop so rapidly that the picture of meningitis is obscured or does not have time to develop before the patient dies

In the past two decades antibiotics and chemotherapy have greatly reduced the over-all mortality as well as the frequency of secondary cases. If proper therapy is promptly initiated, complete recovery can be expected in a high percentage of cases Rarely, deafness, impaired vision, or a paresis will persist.

Tuberculous Meningitis

Secondary to tuberculosis elsewhere, this begins insidiously with malaise, restlessness, irritability, and unexplained fever or headache The primary tuberculous focus may or may not be found Within a week or so, stiff neck, Kernig and Brudzinski signs appear, followed by indications of increased intracranial pressure, cranial nerve palsies and other focal signs, and eventually convulsions, stupor and coma The spinal fluid is under increased pressure, is clear or has a ground-glass appearance, and, after standing, shows a delicate web-like clot Protein content is increased, sugar and chloride, diminished. Cell count is increased to 25–500 per cu mm with lymphocytes predominating If diligently searched for, tubercle bacilli may be found on stained smear of the centrifuged sediment or pellicle, but often the diagnosis can be confirmed only by repeated guinea pig inoculation or culture of the fluid

Until recently, the disease was uniformly fatal but within the past few years the newer therapeutic agents, when started early and assiduously administered for a long period, have effected cures in a considerable proportion of cases. In spite of adequate treatment some patients are left with deafness, blindness, other permanent focal change, or some degree of mental impairment.

Benign Lymphocytic Meningitis

Also known as *acute lymphocytic choriomeningitis*, this is usually preceded by the signs of a mild to moderate upper respiratory infection of 1–3 weeks' duration Several strains of virus can produce the disease. Moderate fever, headache, stiff neck, and perhaps photophobia herald the onset of meningeal involvement Kernig's sign, and depression or accentuation of reflexes may develop. Leukocytosis is the rule The spinal fluid shows increased pressure, elevated protein, normal sugar, and 500–1000 or more cells per cu mm, with lymphocytes predominating. After 2–3 weeks during which the patient does not appear extremely ill, gradual recovery without residuals ensues.

Epidural Abscess

Due to extension of a focus of osteomyelitis of the skull secondary to otitis media, mastoiditis, sinusitis, or external injury, this is marked by high fever, headache, local tenderness, evidence of increased intracranial pressure, and perhaps focal signs The spinal fluid shows increased pressure with or without elevation of protein or cell count Without operation epidural abscess can rarely be differentiated from brain abscess.

Subdural Abscess

Also know as *cerebral subdural empyema*, this lesion can develop from the same causes as epidural abscess In addition to symptoms and signs referable to the underlying trouble, one finds increasing fever, headache, stiffness of neck, perhaps Brudzinski's or Kernig's sign and, later, focal or generalized seizures and focal signs such as hemiparesis, aphasia, or cortical sensory loss The spinal fluid shows elevated pressure and protein, normal sugar, and 20–5000 cells per cu mm, usually with polymorphonuclear leukocytes predominating

Neurosyphilis

Any person with known syphilis, a history of syphilis, or positive serologic and/or T P I tests is to be regarded as a candidate for neurosyphilis To exclude this form of the disease, he must have one or more examinations of his spinal fluid *(see below)*

Since *Treponema pallidum* can invade any or all of the tissues of the central nervous system, it can create clinical syndromes which reflect predominant involvement of the meninges, vessels, or parenchyma of the brain or cord As a rule, the patterns are well defined, but overlapping sometimes occurs They are described as a group in this section because it is believed that the organisms first attack the meninges and spread from them to involve the blood vessels and parenchyma Clinical manifestations of meningeal and vascular disease appear within a few months to years after the primary infection, whereas those of parenchymatous involvement rarely develop until 10–20 years later.

Asymptomatic Neurosyphilis This term is applied to cases in which there are no clinical symptoms or signs of central nervous system involvement but in which the spinal fluid shows characteristic alterations—elevated protein, 10 or more lymphocytes per cu mm, positive complement fixation or flocculation test, and abnormal colloidal gold curve Clinical evidence of syphilis elsewhere may or may not be present, serologic tests are usually but not necesarily positive. To detect asymptomatic neurosyphilis and initiate treatment before the advent of clinical changes, it is imperative that the spinal fluid be examined in every person with suspected or proved syphilis Furthermore, once a supposed adequate course of therapy has been completed, it is equally imperative that the fluid be re-examined at intervals for several years in order to exclude subsequent central nervous system involvement or relapse

Meningeal Neurosyphilis. As a rule, manifestations appear within 2 years after the primary infection. They tend to be predominantly cerebral or spinal

but may reflect damage to both areas. The picture varies with the acuteness and site of the process. In most cases of cerebral meningeal involvement, onset is gradual with headache, often worse at night and sometimes paroxysmal. Disease at the base of the brain is marked by cranial nerve disturbances; over the vortex, by focal cortical signs such as hemiplegia, aphasia, or seizures. Severe headache, vomiting, and papilledema usually develop from increased intracranial pressure due to interference with free flow of cerebrospinal fluid by the inflammatory reaction. A small percentage of cases show the classic signs [illegible] the patient, whereupon early detection and treatment may [illegible]. Spinal meningeal involvement is indicated by pain, paresthesia, and perhaps sensory or motor impairment following nerve root distribution.

With proper treatment recovery from syphilitic meningitis is to be expected. If the patient is not adequately treated, spontaneous remission is probable but he may be left with some permanent nerve or focal cerebral damage; subsequent development of vascular or parenchymal disease is likely.

Gumma of the meninges, which is rare, creates signs of brain or spinal cord tumor. It will not respond to antisyphilitic therapy alone. If the lesion is surgically accessible, favorable results may be obtained by a combination of medical and surgical treatment.

Meningovascular Syphilis. Syphilitic arteritis of the central nervous system rarely exists without associated meningitis. From the clinical standpoint meningovascular syphilis refers essentially to cases in which the process in one or more arteries has progressed to the point of thrombosis with infarction. Although an episode may occur at any time, it is most apt to appear within 2–8 years after the primary infection. When the brain is involved, the picture is similar to that of arterial occlusion from other cause. Prodromal symptoms such as headache, irritability, emotional lability, transient paresthesias or pareses often precede the major insult. Occlusion of a major spinal vessel starts abruptly with flaccid paraplegia and sensory loss below the level of the lesion, and impaired function of bladder and rectum. In the older age group with arteriosclerosis and hypertension one can distinguish between a syphilitic and arteriosclerotic vascular accident only by blood and spinal fluid findings. As in occlusion from other causes, prognosis depends on the size of the vessel involved; in general, the outlook for functional improvement of impaired parts is better than in arteriosclerosis. Although antiluetic treatment may have no appreciable effect on the established lesion, it is indicated to prevent further thromboses and later development of parenchymatous disease.

Tabes Dorsalis. Signs and symptoms reflecting degenerative changes in the posterior roots, posterior columns of the cord, and sometimes the brain stem appear within 10–20 years after the primary infection, occasionally earlier or later.

The classic symptoms, any of which may be the first to develop, are stumbling or uncertainty in walking, especially in the dark, compressive or squeezing pains

in the abdomen or thorax (*girdle pains*), paresthesias of the extremities, bouts of severe sharp shooting or darting pains in the lower, sometimes upper, extremities (*lightning pains*), recurrent episodes of intense abdominal pain appearing and disappearing suddenly and accompanied by retching and vomiting (*gastric crises*), difficulty in urination, and double or impaired vision. Anosmia or uni- or bilateral deafness sometimes occurs In the advanced case one may also find urinary retention with overflow incontinence, loss of libido, impotence, or constipation due to impaired evacuation mechanism. An occasional patient will experience bouts of severe bladder pain with urgency, rectal pain, or throat pain with dyspnea, stertor and cyanosis (*bladder, rectal* or *laryngeal crises*)

The important physical signs are Argyll-Robertson or other pupillary variant (*see* Chap 4), Romberg sign, ataxic slapping gait and other indications of impaired position sense, absence of vibratory sensation predominant in the lower extremities, scattered areas of hypalgesia and hypesthesia of the trunk and extremities, and diminished to absent ankle and knee jerks. If the cervical cord is involved, ataxia of the upper extremities will be indicated by clumsiness of finer movements, such as buttoning or writing Lid ptosis, ophthalmoplegia, visual field defect, and optic atrophy may be observed, the last-named, although usually found in tabes or taboparesis, sometimes occurs independently as a manifestation of neurosyphilis. Atony, diminished pain sensation, and perhaps atrophy of muscles in affected regions may be found A small number of patients will eventually develop one or more Charcot joints (*see* Chap. 35), most likely of the knee, ankle, spine, or small joints of the foot, or a perforating ulcer of the foot (*see* Chap. 31)

Serologic tests are positive in the early cases but may be negative in the long-standing ones Typically, the spinal fluid shows a positive complement fixation or flocculation test, slight to moderate lymphocytosis, increased protein, and abnormal, usually mid-zone, colloidal gold response With time these variants gradually become less pronounced In the spontaneously arrested or adequately treated case, the fluid may be normal

Tabes dorsalis is often self-limited (*burnt-out tabes*) The patient will have some combination of the symptoms and signs just described but will develop no new ones Other untreated cases show slow but relentless progression with increasing incapacity, harassing pains, malnutrition, and ultimate death from intercurrent infection, most likely of the urinary tract Adequate therapy will prevent further advance and effect some symptomatic improvement, but when the clinical manifestations are well established before treatment is initiated, irreversible changes are reflected by persistence of such symptoms and signs as lightning pains, gastric crises, ataxia, bladder disturbances, visual impairment, and Charcot joints.

Dementia Paralytica. Also known as *general paresis*, this is due to spirochetal meningoencephalitis, which causes atrophy and impaired function of the cerebral cortex. Clinical onset, usually occurring 10–20 years after the primary infection, is marked by insidiously developing changes in character, memory, or judgment The patient's family or associates may notice irritability or belliger-

ence, forgetfulness, carelessness in dress or money matters, lack of decision, loss of business acumen, or depressive or hypochondriacal trends. Sometimes he himself will complain of fatigue, headaches, insomnia, forgetfulness, or difficulty in concentration. At this stage the diagnosis of psychoneurosis is apt to be erroneously made. Within months the picture becomes that of a definite psychosis. The most common type is simple dementia, characterized by confusion, apathy, loss of memory, poor judgment, disorientation, and lack of insight and power of calculation. Next most common is the grandiose form, marked especially by euphoria, expansiveness, overactivity, delusions of grandeur, and megalomania. In other cases, paranoia, depression, or complaints suggesting hypochondriasis predominate. Sometimes the psychosis begins abruptly without prodromal signals. Seizures, transient mono- or hemiplegia, or transient aphasia may appear; occasionally such a focal episode occurs as the first sign and is followed weeks to months later by psychotic behavior.

Typical physical findings are irregular tremors of the face, tongue, and hands, pupillary variants of one form or another, and exaggerated deep reflexes with normal plantar responses. Tremulous to slurred speech, at first brought out only by test phrases but later evident in ordinary conversation, and tremulous, scrawly handwriting are characteristic. Generalized muscular weakness eventually develops, but frank permanent paralysis is uncommon. The sensory system is rarely impaired.

In a small number of cases general paresis and tabes occur together (*taboparesis*). Here, in addition to the usual manifestations of paresis, one finds absence of deep reflexes and other indications of spinal cord and root involvement as described in the preceding section.

Serologic tests for syphilis are almost invariably positive. The spinal fluid is abnormal in all untreated cases—normal pressure, increased protein, 15-100 cells per cu. mm. (mostly lymphocytes), abnormal, usually first-zone, colloidal gold curve, and positive complement fixation and flocculation tests.

The untreated case progresses within months to years to the point of complete mental and physical disintegration. The patient becomes totally demented, bedridden, unable to care for himself, and ultimately dies of malnutrition and intercurrent infection. The response to therapy is dependent on the stage at which the disease is diagnosed and treated. In the very early case, decided improvement in mental and physical status may be obtained. Later, progression can be halted and some improvement expected, but irreversible parenchymal changes make total recovery impossible.

THE NEUROSES

Under this heading are included a number of common disorders, which are often confused with the various medical and neurological disturbances described in the preceding pages. One has the clinical impression that they are more likely to be found where there is a family history of emotional instability and in patients who show some degree of autonomic nervous system instability, such as ready blushing and paling of the skin, increased sweating, coldness of

hands, tachycardia, sighing respirations, tremor, and gastro-intestinal disturbances, especially anorexia, aerophagy, gaseous eructations and constipation or diarrhea. Unhappy or insecure childhood is often in the background. The borderlines between the various types of neurosis are not always clearly defined; many persons show indications of more than one Furthermore, it is sometimes difficult to distinguish between a neurosis and a mild depressive or other form of psychosis.

In this complex field distinction must be drawn between the patient who repeatedly or continuously overreacts with neurotic symptoms to situations which by ordinary standards should be handled with equanimity and the one who temporarily develops such symptoms in response to overwhelming or unusual emotional stress or fatigue For example, the nervous, readily disturbed father who becomes emotionally ill even to the point of hysteria over the minor sickness of his child or some fancied derogatory, but actually inconsequential, remark made by his wife or employer belongs in a different category from the soldier with a good performance record who suddenly "cracks" after 3 weeks in a foxhole. The former, being unstable and having a low threshold of emotional endurance, can rightly be classed as neurotic. The latter, fundamentally stable, develops his illness as a result of major stress His acute disturbance is a response to a situation which might be expected to create trouble in all but the exceptionally imperturbable Given escape from the situation or time to adjust, he can be expected to recover without persistence of symptoms attributable to inner unrest or turmoil

Anxiety Neurosis

Acute Anxiety Attack. Acute episodes of panic can develop in the normal person under circumstances of exceptional stress but are more frequently encountered, often repeatedly, in patients with some underlying emotional disturbance, especially chronic anxiety state, hysteria, and agitated depression In the latter group attacks occur without any obvious provocation or in response to some minor disquieting factor They are particularly likely to start when the patient is in a crowd, elevator, or other place where he feels shut in but in some cases they will awaken him from sleep Beginning suddenly, an episode is marked by intense fear, a feeling of choking, breathlessness or smothering, palpitation, pain in the chest, giddiness, trembling, and sweating The apprehension may not be directed at anything particular or be specific, such as fear of death, the heart's stopping, losing the mind, falling in the street, fainting in church, or other comparable catastrophe Physical examination is non-contributory except for showing such evidences of panic as obvious tenseness, flushing, sweating, tremulousness, tachycardia, hyperventilation, or sighing respirations. Depending on the circumstances under which they occur, attacks last for minutes to days, between them the patient reverts to his usual status.

Chronic Anxiety State. This is a protracted illness, often but not necessarily triggered by a disturbing experience, in which the patient is beset by general

nervousness, *insomnia*, irritability, tremulousness, fatigability, faintness or dizziness, headache, breathlessness, precordial or chest pain, anorexia, *indigestion*, constipation or diarrhea, and unsatisfactory sexual adjustment. As a rule, he has a fear of crowds, theaters, elevators, or other closed-in places. Repeated attacks of acute anxiety are likely to be superimposed, these can occur spontaneously or be precipitated by some incident which is upsetting to the patient but would be inconsequential to the well person. The illness may be incapacitating; remissions and exacerbations are likely. Frequent visits to one or more physicians are the rule. Sighing respirations, mild tremor, tachycardia, and cold, moist hands are common findings, but no evidence of any serious physical defect is detectable. The patient often attributes his illness to "low blood pressure", "anemia", "virus", "acidity", or "gas".

Anxiety manifestations are also common in the other neuroses.

Neurosis with Phobia

This appears to be an exaggerated form of anxiety neurosis, but anxiety attacks are not the presenting feature. The patient has an overwhelming, deeply rooted fear of cancer or other serious malady, enclosed spaces, doing certain things such as riding on a train, walking alone in the street, touching objects because of "germs", or going near a horse, dog, or some object such as a knife. The apprehensiveness can be so profound as totally to incapacitate him for ordinary living.

Compulsion Neurosis

Here one finds compulsions and obsessions, extreme indecision, worry following every decision, and various anxiety symptoms. Typical compulsions are repeated washing of the hands, elaborate ritual in dressing in the morning, numerous trips about the house before retiring to make sure that certain doors and windows are locked and others open to exactly the correct degree, faucets closed, and various articles of clothing and furniture precisely placed. Kleptomania, pyromania, and dypsomania are compulsive phenomena. Common obsessions are thoughts of a profane or blasphemous nature and about death or doing bodily harm to someone.

Hysteria

This predominates in women. Past history often reveals emotional, social, and school problems in childhood, sleep-walking or sleep-talking, and, perhaps, repeated hospitalizations and fruitless operations. Complaints frequently encountered are transient aphonia, blindness or inability to open the eyes, lump in the throat (*globus hystericus*), palpitation, smothering, gastro-intestinal upsets especially episodes of persistent vomiting, and menstrual disturbances. Sensation is apt to be impaired in some part of the body but examination shows the defect to correspond to a functional, not a neuroanatomic, zone. For example, hypesthesia or anesthesia will be found on a leg or foot (*stocking anesthesia*), arm or hand (*glove anesthesia*), or one entire side of the body. The pa-

tient may be unable to use a limb, but the spasticity or flaccidity and abnormal reflexes associated with true paralysis are not found. Unusual "*trances*", attacks tending to mimic convulsive seizures, and episodes of emotional outburst with screaming, weeping, laughing, and thrashing about are common. Memory shows wide gaps Ignorance of sex matters prior to adulthood or marriage, profession of disinterest in the subject, frigidity or dyspareunia, and imagined rape are often elicited in the history.

The illness is episodic. Exacerbations are likely to be precipitated by real or fancied disturbing occurrences of a minor nature; the emotional outbursts and seizure-like attacks occur when onlookers are present The patient is sometimes peculiarly nonchalant about his illness and may even smile throughout interviews with the physician Often one will find that he, consciously or unconsciously, has a gain to derive from the sickness—constant attention from spouse, children or relatives, freedom from household or other responsibilities, exemption from military service, insurance compensation.

Hypochondriasis

An ill-defined entity on the borderline between neurosis and psychosis, this is marked by insistent and almost delusional preoccupations centered about bodily functions and submerging all other interests The patient will discuss nothing but his ailments He characteristically flits from one doctor to another and in the interim doses himself with nostrums of his own prescribing, patronizes quacks, and experiments with current "health" fads. Here, too, there is apt to be a history of repeated hospitalizations and, perhaps, operations which proved to be unnecessary or ill-advised Repeated studies fail to disclose any systemic or local variants to account for the manifold complaints The patient is unwilling to accept the explanation that his illness is of psychogenic origin and often appears to derive satisfaction from regarding himself as a "baffling case"

THE PSYCHOSES

In general, the psychoses are more serious than the neuroses and often necessitate treatment in a mental hospital

Toxic Psychosis

Also known as *delirium*, this is encountered as a complication of any serious febrile disease, heart failure, anemia, uremia, thyrotoxicosis, vitamin deficiency, and other disorders, and during use or withdrawal of alcohol or certain drugs, especially opiates, bromides, barbiturates, amphetamine, cocaine, meperidine, atropine, scopolamine, ACTH, and corticosteroids Many old people are peculiarly sensitive to ordinary therapeutic doses of morphine and other narcotics A comparable picture can be found in the postoperative, postpartum or post-traumatic state and in diseases of the central nervous system such as encephalitis, meningitis, and brain tumor, trauma, abscess or vascular accident. Among the common signs are clouded consciousness, disorientation, rest-

lessness, agitation, muttering, nightmare-like states, hallucinations, delusions, carphologia, tremors, incoordination of fine movements, and slurring of speech. The hallucinations and delusions are of the fearful type; for example, the patient is likely to see animals in the room or think that the medicine offered him is poison. Sometimes his fear may prompt him to escape by jumping from a window, totally without warning. Mental status returns to normal within a few days to 1-2 weeks after recovery from the underlying trouble.

In *bromide poisoning*, the patient may be dull, sluggish or stuporous, or become delirious, frequently with severe hallucinosis and, sometimes, accompanying neurologic signs such as poorly reacting or unequal pupils, tremors, and hyperactive reflexes. Often the latter picture will develop during the withdrawal period even though the dosage is slowly diminished. Detection of a high concentration of bromides in the blood is confirmatory.

The manifestations of *barbituate intoxication* can vary from confusion, slurred speech, ataxia and nystagmus to severe delirium, stupor, or coma with respiratory depression, diminished to absent reflexes and signs of peripheral circulatory failure. Determination of barbituate blood level is important. In chronic addiction, too rapid withdrawal sometimes causes generalized seizures.

Whenever one encounters mental or neurologic signs which appear bizarre or do not conform to the usual patterns, bromide, barbituate or other drug intoxication must be excluded. Denial by the patient, and often by his relatives, of the use of any such agent is totally untrustworthy.

Alcoholic Psychosis

Chronic alcoholism engenders serious nervous system disturbances by creating (1) a nutritional deficiency presumably from failure to eat or poor absorption, or (2) changes attributable to relative or total withdrawal following long habituation. Direct toxic effect of alcohol *per se* is not regarded as a factor. Although the psychiatric and neurologic manifestations are numerous and protean, only the more commonly encountered syndromes are described. These sometimes occur in pure form but overlapping is frequent, symptoms and signs characteristic of more than one often develop in the same patient. Even though some show only minimal psychotic aberrations, they are discussed here for convenience.

Alcoholic Tremulousness. Popularly (or unpopularly) known as the *jitters*, *shakes* or *heebie jeebies*, this term is applied by Victor and Adams (Raymond D.)[1] to a syndrome usually encountered in the periodic drinker a day or so after the end of a bout. It can also occur in the constant drinker if his customary daily ration is appreciably reduced or, for a while each morning, as a result of deprivation during sleep, even though he drinks all day. Nervousness, shakiness, restlessness, agitation, inability to concentrate, anorexia, nausea and vomiting are the outstanding features. As a rule, they appear first in the morning and for a few days can be controlled by prompt resumption of drinking. Soon this form of self-prescribed therapy is no longer effective and for one reason or another—nausea and vomiting, self-disgust, or cut-off of liquor supply—the patient stops drinking, whereupon his symptoms all become more pronounced. He becomes

more perturbed and irritable, fearful, and shows a constant, irregular, fine to gross tremor which becomes accentuated on activity or any added emotional stress Insomnia is striking At this stage he is usually lucid, well oriented, and aware of the nature of his sickness. As the misery progresses, false perceptions, varying from nightmarish experiences during periods of characteristically fretful sleep to definite hallucinations, develop in about one quarter of cases Even when these are present, the patient remains in contact and with his sensorium otherwise relatively unimpaired.

As a rule, the more striking changes will disappear within a week or so but inner restlessness, jumpiness, some tremulousness and feelings of insecurity may persist for an appreciably longer period Some cases proceed into a state of delirium tremens.

Delirium Tremens. This is the most serious complication of reduction or withdrawal in the *constant or periodic heavy drinker It may develop following* a period of tremulousness, hallucinosis or seizures Often it appears while the patient is in a hospital for treatment of a major illness, operation, or injury He becomes agitated, frightened, overactive often to the point of requiring *restraint, garrulous, tremulous, and constantly picks at the bedclothes. He is* badly confused, totally disoriented as to time and place, hallucinated, and misidentifies and misinterprets people and objects in his room Fearfulness attributable to delusions and hallucinations is pronounced Dilated pupils, fever, tachycardia, and excessive sweating are usually found

The most common hallucinations are visual but auditory, olfactory or mixed types are sometimes experienced The first named, almost always terrifying, center around humans, animals and insects, which often appear in bizarre, distorted and horrifying forms. The auditory phenomena take the form of voices, usually of relatives or acquaintances and of critical, defamatory, or even threatening import. They can be heard coming from the closet, through a wall, or even over the radio and may address the patient directly or be speaking about him In his mind, they are so vivid that he will react, for example, by seeking some *form of protection from the threats or even attempting* suicide to escape the derogatory accusations If, as sometimes happens, the voices bring inconsequential messages, he shows little concern

In delirium tremens the prognosis is usually good Most patients recover within 4 days, some, only after 2–4 weeks After the episode, symptoms and signs of Wernicke-Korsakoff syndrome may be discovered The gravity of the situation is greatly enhanced by the presence of any concomitant disease or injury In some cases, death results from intercurrent infection, in others, the cause is not understood. It may be preceded by peripheral circulatory failure or occur abruptly, without warning.

Auditory Hallucinosis. This sometimes appears in pure form with otherwise clear mentation. The predominant symptom is the hearing of voices as described above—almost always threatening and so ominous as to throw the patient into an overwhelming panic. Because of suicidal or homicidal trends based on his hallucinations, he must be hospitalized and closely watched This illness usually terminates within a few days. However, in an occasional case the audi-

tory hallucinations will persist for weeks to months, because of them, the patient becomes moody, depressed, withdrawn, and may even develop a picture simulating schizophrenia

Alcoholic Seizures. Also encountered only in those who have been heavy drinkers for many years, these develop within a few hours to days *following relative to total reduction of alcoholic intake.* They occur *multiply—2–6 in 21–48* hours—and take the form of generalized convulsions with loss of consciousness. They are often followed by delirium tremens but are rarely observed during its course. As a rule they appear only during the relatively short withdrawal period and do not recur between times.

Before one can be certain that seizures in an alcoholic are a reflection of his addiction, careful investigation is essential to exclude other possible causes. Since alcoholic epilepsy begins only after years of drinking, it appears in the same age group in which seizures created by brain tumor, cerebral vascular disease, and neurosyphilis are also most apt to be encountered. Theoretically, idiopathic epilepsy, which usually begins in youth, should be easily distinguishable from the alcoholic type but actually differentiation is not always easy because the former can be accentuated or, when latent, perhaps brought out by heavy drinking The inebriate is often unaware of a head injury acquired while intoxicated For this reason, it is possible erroneously to attribute seizures to alcoholism when actually they are of traumatic origin, especially since the latter, too, can be accentuated by chronic overindulgence Focal seizures must never be attributed to alcoholism.

Wernicke-Korsakoff Syndrome. Although Wernicke's disease and Korsakoff's psychosis are usually discussed separately, we combine them, in accordance with the teaching of Victor and Adams[1], who believe that these represent different facets of the same pathologic process. Since they are attributed to malnutrition, they can be encountered in a variety of circumstances, but are included in this section because of their relative frequency in chronic alcoholics.

Wernicke's disease is marked by the abrupt appearance of mental changes, ocular motor palsies, and ataxia The psychic disturbance may take several forms apathy, inattention, lack of concentration, and disorientation; delirium resembling that of delirium tremens, impairment of memory as described below The eyes show horizontal and vertical nystagmus, loss of conjugate gaze, and paresis of the external rectus muscles with strabismus and diplopia In the extreme case one may find complete paralysis of ocular movement and constricted, non-reacting pupils The ataxia, which chiefly involves gait, varies from slight uncertainty detected on special tests to inability to walk or stand without assistance It is thought to be of cerebellar origin. Mild polyneuropathy is sometimes found, it is not sufficient to account for the ataxia. Prompt recovery can usually be effected by adequate therapy with vitamins and other nutritional elements

In the alcoholic, most cases of *Korsakoff's psychosis* are preceded by mild to severe manifestations of Wernicke's disease. Its outstanding feature is a defect in which recent memory and retention are lost to a degree out of proportion to impairment of other intellectual faculties. Often unapparent in a cursory

examination, the deficiency may be brought out by careful testing—the patient is unable to perform simple tests, presumably because he cannot retain instructions long enough to carry them out. Other features are confusion, inability to think clearly, and confabulation—filling-in of memory gaps by fabrication. The last named, contrary to generally accepted teaching, is neither always present nor a requisite for the diagnosis. Polyneuropathy may or may not be present, with proper treatment, this will usually clear entirely. Psychiatrically, the patient may recover completely, partially, or not at all. A mental picture similar to that of Korsakoff's psychosis can be created by tumor or trauma involving the walls and floor of the third ventricle.

Schizophrenia (Dementia Praecox)

This serious disorder usually develops in young adults. Early manifestations are lack of concentration, withdrawal from customary activities, vague day-dreaming, apathy, suspiciousness, hallucinations, delusions, and ideas of reference (*i.e.*, the patient thinks all occurrences are related to himself). Thoughts of being influenced by electricity or hypnotism, and religious ideas and preoccupations concerned with such matters as salvation, punishment, and resurrection are common. The latter are sometimes difficult to differentiate from deep religious feeling in a normal person. Rambling or scattered speech often with neologisms, blocking, and impulsive behavior are characteristic. The course is usually progressive, with minor temporary remissions.

Simple schizophrenia is characterized by deterioration of attention and discrimination; the patient appears to become less attentive and increasingly stupid. *Hebephrenia* shows slovenliness, silly talk and bizarre behavior; *catatonic schizophrenia*, phases with excitement or maniacal behavior, and phases with maintenance of curious posture, staring, catalepsy, negativism, mutism, and stupor, *paranoid schizophrenia*, ideas of reference, misinterpretations, hallucinations, and delusions of persecution. *The paranoid patient is often a dangerous homicidal risk.*

Manic-Depressive Psychosis

A family background of "nervous breakdowns", suicides, or frank manic-depressive psychosis is common. Manic or depressive attacks or both, lasting from several weeks to years but with intervals of relatively normal status, feature this syndrome. The general make-up of the manic-depressive person is fairly adequate; between psychotic cycles good occupational and social adjustment is the rule.

In the *hypomanic* state, the patient is overactive and overtalkative, constantly "on the go", aggressive, and to casual observation would seem merely a "live wire". Spells of useless or extravagant spending, dispatching of wordy telegrams, frequent unjustified long-distance telephone calls, and concoction of grandiose schemes are typical. Sexual overactivity is frequent; young women previously chaste may become promiscuous. The patient is good-natured until thwarted, whereupon he becomes angry, suspicious, and contentious. Before

his illness is recognized, he may lose all his money, suffer the consequences of sexual irregularities, or become involved in other serious difficulties. Excessive drinking is often a symptom of hypo- or overt mania and adds fuel to the fire; in the hypomanic it may obscure the underlying trouble. When the patient is normal or depressed, alcoholism is not ordinarily a problem.

In the *manic* state, there is even greater overactivity, incessant talking, flight of ideas, rhyming, punning, and easy distractibility The patient has delusions of self-exaltation, identifying himself with prominent personages or even God; the urge of self-assertion is tremendous; opposition is met with impatience, belligerence, or violence.

In the *depressed* phase, one finds inactivity, feeling of inertia or exhaustion, slow, infrequent speech, lonesomeness, hopelessness, and despair Insomnia, anorexia, weight-loss, amenorrhea, and constipation are accompanying symptoms Self-accusation, feelings of guilt, and a conviction that he or his family have been singled out for punishment obsess the patient; he may even describe the exact form the punishment will take *Suicide is a decided hazard.*

Agitated Depression

Although sometimes called *involutional melancholia*, and usually appearing within the fifth or sixth decade, this is not necessarily directly associated with the menopause The patient is worried to dejected, listless or overactive, uneasy, agitated, panicky, and often feels a threat of impending poverty, ruin, or incapacitating illness He is beset by hypochondriacal symptoms Agitation compels him to walk up and down the room wringing his hands and muttering such phrases as, "Oh, my God! Oh, my God! There is no use! I can't stand it Why must I suffer so? I wish I were dead" He may hear accusatory voices or anticipate some form of torture These patients are also suicidal risks, but if the anxiety features predominate, the depressive and suicidal aspects may be obscured.

Reference

1 Victor, M., and Adams, R. D *Metabolic and Toxic Diseases of the Nervous System* Vol XXXII, Proceedings of the Association for Research in Nervous and Mental Disease Baltimore, The Williams & Wilkins Company, 1953

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